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A CASE OF GUNSHOT WOUND OF THE BRAIN IN WHICH THE ROENTGEN RAYS SHOWED THE PRESENCE OF EIGHT FRAGMENTS OF THE BULLET; LOCALIZATION BY SWEET'S METHOD MADE OPERATION INADVISABLE.

WITH A DESCRIPTION OF AN IMPROVED APPARATUS FOR LOCALIZATION OF FOREIGN BODIES.

By W. W. Keen, M.D.,

AND

Wm. M. Sweet, M.D.,

OF PHILADELPHIA.

DR. KEEN'S REPORT OF THE CASE.

The following case is put on record as an excellent illustration of the inadvisability at times of removing a foreign body from the brain, and also of the probable rebound of a ball from the inner surface of the skull:

Clarence P., aged fifteen years, of Victoria, B. C., first consulted me on April 9, 1903, at the instance of Dr. O. M. Jones, of Victoria, and of Sir Victor Horsley.

On October 24, 1902, he accidentally shot himself with a 22-calibre rifle. The bullet entered the right side of the forehead a half-inch above the eyebrow at the junction of the middle and external thirds. Dr. Jones saw him half an hour after the accident occurred, and found brain matter and blood oozing from the wound. The boy was collapsed and complained of numbness in the left leg and foot, but could move them. He was taken to a hospital, anæsthetized, the wound enlarged slightly, thoroughly cleansed, and an antiseptic dressing applied. The opening in the bone readily admitted the tip of the forefinger. Several small splinters of bone were removed, and from under the skin, about one inch *above* the opening in the bone, a sliver

of the bullet was removed. The weight of a similar unused bullet is 29½ grains. The sliver weighed 5½ grains. The eight remaining pieces together, therefore, weigh 24 grains; the largest piece prob-

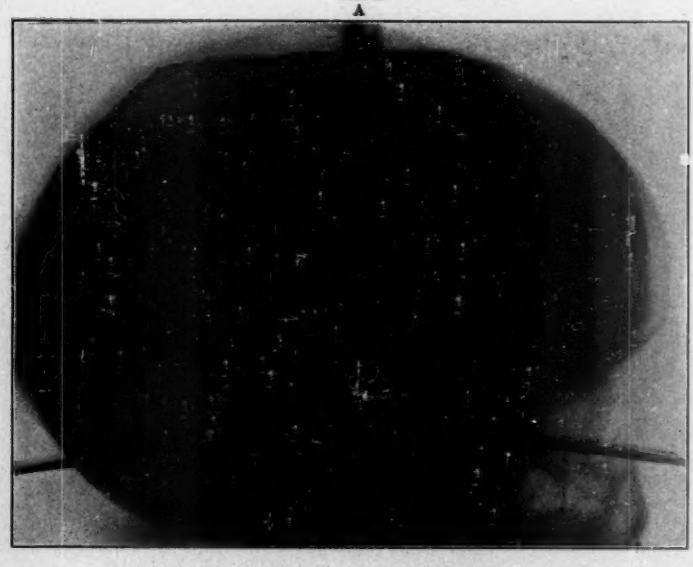
FIG. 1.

The upper figure is that of a similar bullet to the one causing the accident (weight 29½ grains); the lower figure is the sliver removed from under the skin above the wound of entrance (weight, 5½ grains). Both are natural size.

ably weighed about twenty grains (Fig. 1). Dr. Jones, wisely, did not probe for the bullet.

At the end of two or three days after the accident the left leg became entirely paralyzed. The first night he was extremely restless,

FIG. 2.
A



Skiagraph showing the eight fragments of the bullet.

with frequent involuntary movements of the right leg and arm, and on the day after the accident he had half a dozen very severe epileptiform convulsions, which ceased after a few doses of chloral and

bromide and have not recurred. He gradually improved, never lost his intelligence, the paralysis diminished, and in the course of three months he was able to get about.

Status Præsens. April 9, 1903. There is a small scar just above the right eyebrow. No perceptible aperture in the bone can be felt. There is a little weakness still left in the left leg and foot, as shown by the fact that he balances himself unsteadily upon that foot, whereas upon the right one his balance is good. There is also some increased knee-jerk in the left leg, both with and without reinforcement.

The patient's friends in England had consulted both Sir Frederick Treves and Sir Victor Horsley in reference to his treatment. A brief synopsis of their opinions is as follows: That the position of the retained bullet should be accurately determined; that the ball is now not likely to shift its position; that it is a possible source of future irritation which might result in traumatic epilepsy, and that the advisability of the removal of the ball would depend upon its location.

Dr. Sweet then made some skiagraphs of the head, of which one is presented in Fig. 2. By his method of localization he determined the following facts:

In the brain there are one principal fragment and seven other minute fragments of the bullet, the largest of the latter being about the size of a medium-sized pin's head. Two of these fragments are at the point of entrance. Five of them are scattered through the brain 2.5 cm. to 3 cm. *above* a line running from the wound of entrance to the present position of the ball. The position of each one of these fragments was precisely indicated on the scalp by Dr. Sweet.

After careful consideration of the present location of the bullet and of the boy's excellent recovery, I decided that it would be inadvisable to remove the ball. Of course, the removal of the smaller fragments was not even considered. The following were the reasons for my decision: The ball lay vertically 5 cm. below a point 2.2 cm. to the right of the middle line and 1.5 cm. back of the fissure of Rolando on a line parallel with the middle line (Fig. 3). Dr. Sweet marked not only the point on the top of the head corresponding vertically to the ball, but also a point above the ear which corresponded to it horizontally. This point was, of course, 5 cm. below the level of the top of the head, but on the curved surface of the scalp was 8.3 cm. from the middle line. It was 4.5 cm. back of the fissure of Rolando. In the horizontal plane from the surface of the head toward the middle line the ball lay at a distance of 6.3 cm. (Fig. 4). As it was very important that these two points corresponding horizontally and vertically to the position of the ball should be accurately and permanently marked, with a view to an accurate determination in the future whether any change in the position of the ball had taken place, they were marked in the following way: At the points which Dr. Sweet had marked upon the shaven scalp I injected a few drops of cocaine and then excised a small round piece of scalp, including the hair-follicles, so that there would be two small permanent bald spots at these points. As

the boy was a very tall, well-developed lad, who had attained almost all of his growth, especially of the head, any later growth will not practically invalidate these measurements.

FIG. 3.

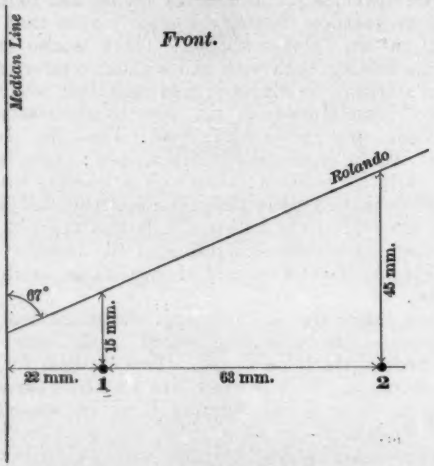
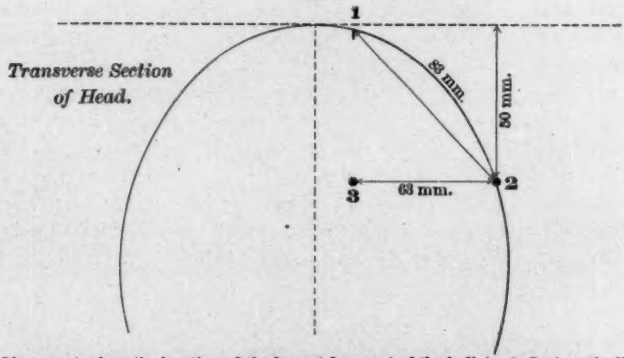


FIG. 4.



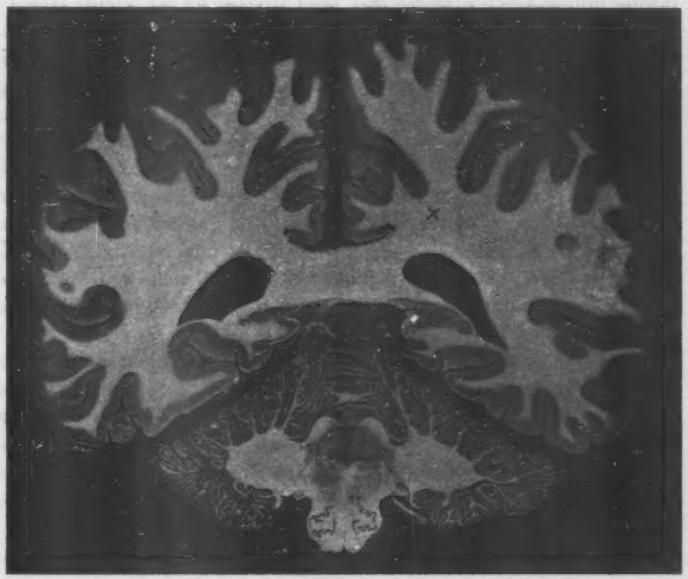
Diagrams to show the location of the largest fragment of the bullet. 1. Spot vertically over ball. 2. Spot horizontally corresponding to ball. 3. Site of ball.
[NOTE.—The measurement 83 mm. should be from the middle line to 2, and not from 1 to 2.]

I wished then to determine the relation of the ball to the lateral ventricle, and for this purpose made a section of a brain hardened in formalin, at a point as nearly as possible corresponding to the figures given above. Comparing this with Dalton's vertical transverse sections through the brain, I found that it corresponded to Plate 15 of series C quite exactly. Applying the foregoing measurements then to this

figure, I determined that the position of the ball corresponded to the cross marked on a reproduction of this plate (Fig. 5). It will be observed that the ball lies far below the cortical leg centre and just above the lateral ventricle.

The course of the bullet was evidently not in a straight line from the wound of entrance to its present position. This is shown by four facts: 1. The boy states that at the time of the accident he was not looking directly into the barrel of the rifle, in which case the antero-posterior axis of the head would be nearly vertical, but that his head

FIG. 5.



X shows the present position of the largest fragment of the bullet.

was only moderately flexed. 2. At the wound of entrance, as the ball penetrated the bone, a considerable sliver was taken off the ball by the *upper* edge of the bone and lay 2.5 cm. above this wound. 3. None of the fragments, except those at the point of entrance, lie in the direct line between the wound of entrance and the present position of the ball; they all lie *above* this line and at a considerable distance above it. 4. If the ball had gone in a straight line from the wound of entrance to its present position, it would not have touched the cortical leg centre at all, though it is possible it might have di-

vided some of the descending fibres from the leg centre. It seems, however, much more likely that the ball passed in a line from the wound of entrance to the point A, Fig. 2, and then was deflected to its present position, and in its course traversed the cortex. It would then have injured precisely the centre for the left leg.

I decided, therefore, that it would be inadvisable to remove the ball. In its present position it is doing no harm to the leg; his paralysis has steadily diminished, and in the course of a few months will probably entirely disappear. Any attempt to remove the bullet would cause very much more injury to the brain than the bullet itself does in its present position. Accordingly I sent the patient home, with the following injunctions: First, as had been advised by Sir Victor Horsley, that he should not begin to study or do much reading until a year had elapsed from the time of the accident, and that he should then study under a tutor for a year, so as to postpone the excitement of a large school for two years from the date of the accident; second, that he should be allowed a greater degree of exercise as his leg improved; and, third, that if either of two symptoms arose he should immediately be brought back to me, viz., in case he developed persistent severe headache or any other symptoms which might suggest impending cerebral abscess; or if he should have a single epileptic attack, I should want to remove the ball immediately. The ball then would be doing more harm than the surgeon, whereas by any present interference the surgeon would do more harm than the ball.

It will be observed that at the time of the accident he had some severe epileptiform attacks, but has had none since. It is not impossible that he may never have any, and it seemed to me wiser to run the risk of a possible future epilepsy than a certain, immediate, and probably very serious damage to the brain by present surgical interference.

DR. SWEET'S REPORT ON THE METHOD OF LOCATING FOREIGN BODIES IN THE BRAIN AND ELSEWHERE BY THE X-RAYS.

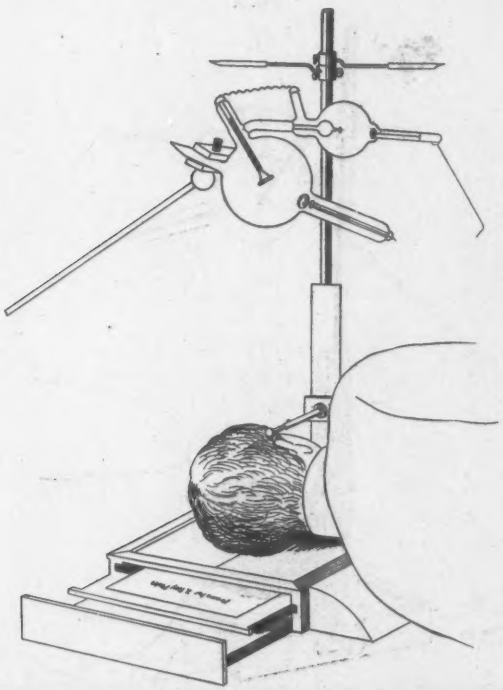
The methods employed in locating foreign bodies by the Roentgen rays are all based upon the triangulation of the planes of shadow of the body, with the X-ray tube in two different positions. Measurements of the distance of the crossing of these planes from one or more points marked upon the skin give the exact situation of the foreign substance.

Accuracy of localization depends upon a knowledge of the position of the tube at the two exposures, its distance from the photographic plate, and upon the proper marking of one or more spots upon the skin by opaque substances that will cast shadows upon the plate. A

special form of apparatus achieves these results in the most satisfactory manner.

The apparatus for locating foreign bodies is similar in principle to that employed so successfully during the past few years in determining the situation of pieces of metal in the eyeball. I have recently designed a new form of localizer, which is shown in the accompanying illustrations. It consists of a firm base, the top of which is covered with sheepskin, and

FIG. 6.



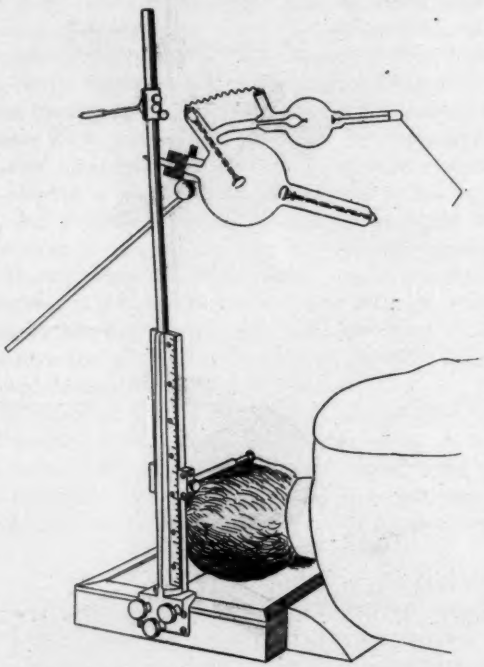
Side view of the indicating apparatus, showing position of tube and indicator, and also receptacle for the photographic plates.

is crossed by two steel wires at right angles to each other. A sliding drawer on one side permits the changing of the photographic plate without disturbing the position of the patient. A hollow upright bar attached to one side of the base supports three movable rods. One rod has its extremity pointed, over which slips an indicating ball. The other rods are employed to indicate the situation of the tube at the two exposures. The centre of the indicating ball is directly above the

crossing of the two wires on the base, and its height is shown by a scale upon the upright bar.

The head or other portion of the body containing the foreign substance to be located rests upon the top of the base portion, the cross-wires, previously inked, leaving a mark upon the skin. The indicating rod is lowered until the ball rests upon the skin, and this spot is also indicated with ink. The distance of the ball above the cross-wires is read from the graduated scale.

FIG. 7.



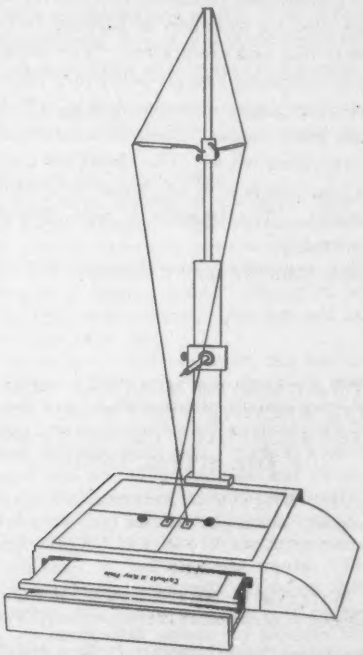
Side view of apparatus, showing indicating rod and graduated upright.

The X-ray tube is placed from eighteen to twenty inches above the plate, and the distance of the anode of the tube carefully measured. One exposure is then made with the tube directly above or to one side of the indicating ball, and a second plate made with the tube from three to four inches from the first position.

After development the plates show the shadows cast by the cross-wires, the indicating ball, and the foreign body. The distance of the foreign substance from the marks upon the skin of the patient may be

determined by plotting upon a flat surface the position of the tube, indicating ball, and the cross-wires at the two exposures, and finding the crossing of the planes of shadow of the foreign body. This method is the one employed in eye-work, and is equally accurate in dealing with other portions of the body, although somewhat more complicated. For this reason I have employed threads to indicate the crossing of the planes of shadow in the new localizing apparatus—a method which has been developed by Mackensie Davidson, of London.

FIG. 8.



Planes of shadow represented by threads, the point of crossing of which indicates the situation of the bullet.

The method of employing the indicating apparatus is as follows: A tracing of the two negatives is made upon a transparent sheet of celluloid, with the point of crossing of the wires corresponding. This celluloid sheet is placed upon the top of the base of the apparatus with the tracing of the shadow of the cross-wires directly above the point of crossing of the wires. The indicating rod is moved to the height it occupied at the time the exposures were made. A thread is now passed from the spot on the celluloid representing the shadow

of the indicating ball at one exposure, touches the point of the indicating rod representing the centre of the ball, and is continued the exact distance that the tube was away from the plate when the radiograph was made. A second thread passes in the same manner from the shadow of the indicating ball at the second exposure. The threads are kept taut by lead weights in the hollow upright tube. These two threads accurately indicate the lines of shadow of the indicating ball at the two exposures, and also the position of the tube. If the ends of the threads resting upon the shadows of the indicating ball are now moved to the spots on the transparent celluloid representing the shadows made by the bullet, their crossing will show its position in the tissues in relation to the indicating ball and cross-wires. The distance of this point of crossing from the indicating ball gives the location of the body in the tissues as measured from the spot on the skin at which the ball rested when the two plates were made. The situation of the bullet may also be measured from the cross-wires. The depth of the body will determine which of the two points is to be chosen in plotting the position of the body for operation. The location of each fragment of the bullet is similarly determined.

The apparatus was made for me by Queen & Co., of Philadelphia.

A REPORT OF TWO CASES OF MULTIPLE SARCOMATOSIS OF THE CENTRAL NERVOUS SYSTEM AND OF ONE CASE OF INTRAMEDULLARY PRIMARY SARCOMA OF THE SPINAL CORD.[1]

By William G. Spiller, M.D.,

Assistant Clinical Professor of Nervous Diseases and Assistant Professor of Neuropathology in the University of Pennsylvania;

and

William F. Hendrickson, M.D.,

Assistant Demonstrator of Pathology in the University of Pennsylvania.

(From the William Pepper Clinical Laboratory, Phoebe A. Hearst Foundation.)

Sarcomatosis of the central nervous system apparently has not received much attention from American physicians,[2] although a few cases have been reported by English writers and quite a number may be found in the German literature. Cases of this character must have occurred in this country, and it is to be hoped that they will be put on record. One such has recently been seen in the service of Dr. F.

[1] Read at the meeting of the American Neurological Association, May 12, 1903.

[2] Dr. H. T. Patrick has called my attention to a case of diffuse sarcoma of the cerebral and spinal pia mater, reported by George K. Weaver in the Journal of Experimental Medicine, 1898, vol. iii., No. 6.

X. Dercum, although the sarcomatosis in this case was not confined to the nervous system. Two cases of sarcomatosis of the brain and spinal cord, or of their membranes, in one of which the sarcomatosis was certainly confined to the central nervous system, and in the other was probably so confined, have been observed by one of us (Spiller), and are reported here in connection with a case of primary sarcoma of the substance of the spinal cord. The first case was to have been reported by the late Dr. W. F. Hendrickson, and the material had been partially studied by him at the time of his death. His notes are in quotation marks.

The patient[1] was in the service of Dr. Van Gasken, at the Woman's Hospital of Philadelphia, and I am much indebted to her for permission to use the clinical notes and to report the case.

CASE I.—L. K., aged twenty-one years, married, was admitted to the Woman's Hospital of Philadelphia, January 4, 1902, when the following notes were made:

The family history was unimportant.

The patient had been strong as a child. She had had scarlatina, diphtheria, and other diseases. She menstruated when fourteen years of age, but her periods were not always regular. They were painless, and lasted from four days to a week. She was well until after she was married, at the age of twenty years. She gave birth to a child five months ago; the labor was normal. She has not had much sore-throat, but last fall her hair came out.

Three days after her child was born she had a severe headache in the occipital region. The pain was shooting, and began at the base of the brain and extended upward; it was very severe but transient. Occasionally she had a sensation of pressure on the head. After the headache passed off she felt giddy and saw everything double, and shortly afterward she was told that she was cross-eyed. She had not had any paralysis of the ocular muscles previously. She had no fever. She had profuse sweating followed by cold, creepy feeling, after which she became very giddy. She had a shooting pain under the sternum. She has been confined to bed thirteen weeks. The toes first began to get stiff, and the stiffness gradually extended upward until now she has no power below the waist. At the first appearance of stiffness of the limbs she suddenly lost power to urinate voluntarily, and since then urination and defecation have been involuntary.

About a week ago a red spot appeared on the hip, and ulceration has occurred at this place.

The tongue is dry and coated.

Pulse, 108; temperature, 98.4° F.; respiration, 28. Two large bed-sores are found on the left thigh.

January 5th. Patient has complained of pain in the back and over the epigastrium.

The result of the examination of the eyes by Dr. Mayo, January 6th, is as follows: Pupils react normally to light and in convergence.

[1] A brief reference to this case was made in a paper published in the University of Pennsylvania Medical Bulletin, April, 1903.

Complete paralysis of the external rectus of each eye with paralysis of one of the oblique muscles, but the condition of the patient renders it impossible to determine which one. Ophthalmoscopic examination: Media clear; marked swelling of optic disks with hazy outlines; vessels tortuous; no hemorrhages; macular region normal.

I saw the patient in consultation January 7, and the result of my examination is as follows:

Pupils are equal and react promptly to light. The inward movement of the left eye in convergence is greater than that of the right. Head is slightly retracted, and neck is somewhat stiff. She has apparently no deafness. A papulopustular eruption is seen on the face and upper parts of the limbs. Tongue is protruded partially; no fibrillary tremor and no atrophy of tongue. No paralysis of facial muscles. Masseter contracts firmly on each side. Headache is general, but more severe in the frontal region; sometimes it is occipital. It is not worse at night. Patient moans continually as if in pain. Sensation to touch, heat, and pain is normal in the face. The head is rotated from side to side slowly and with much pain in the neck.

The grasp of the hands is nearly normal, but the effort to use the hand causes intense pain in the lumbar region. The upper limbs are moved freely in all directions. During the examination the patient yawns frequently. Sensation for touch, heat, and pain in the upper limbs is normal. Biceps tendon, triceps tendon, and wrist reflexes are normal. The muscles of the upper limbs and the ulnar nerves are not tender to pressure. Pressure along the spine is not distinctly painful except between the shoulders.

The lower limbs are completely paralyzed. The patellar reflex on the right side is present but diminished, and on the left side it is almost lost, and on each side the reflex is very little increased by reinforcement. Slight ankle clonus is obtained on the right side, but not on the left. Achilles tendon jerk is exaggerated on the right side, normal on the left. Babinski's reflex is present on the right side, but soon exhausted. It is present also on the left side, and the big toe moves distinctly upward. The plantar reflex is decreased on each side. Sensation for touch, pain, and temperature is apparently abolished in the lower limbs. Muscles and nerves of the lower limbs are without sensation from pressure. Any passive or reflex movements of the lower limbs causes intense pain extending from the front of the abdomen to the back. Sensation to pin-pricks, temperature, and touch is first perceived on the trunk at a level with the seventh rib in the sternal line. The area where anæsthesia passes into normal sensation is not sharply defined. No distinct deformity of the vertebral column is seen.

Cerebro-spinal fluid was obtained by Dr. Everitt in lumbar puncture. It was clear, and without reddish tinge. Fibrin was present in small amount. No cells or bacteria were found. Cultures on agar-agar showed a few colonies. Examination after twenty-four hours' growth showed the presence of a micrococcus. This report was given by Dr. A. W. Peckham.

The patient slept very badly, was restless, and suffered much pain. She died January 22, 1902.

The necropsy was made by Dr. Hendrickson and the following notes are by him:

"*Anatomical Diagnosis.* Multiple tumors of the spinal cord, tumor of cerebellum, congestion of cord and brain; slight congestion of lungs, spleen, liver, and kidneys.

"*Histological Diagnosis.* Small spindle-cell sarcoma of the cerebellum with multiple new-growths of like character of the spinal cord; congestion of heart, spleen, liver, and kidneys.

"*External Appearance.* Body that of a fairly well-developed, but poorly nourished white female. Rigor mortis present in both upper and lower extremities, to a slight degree, although body is still warm. No œdema. Slight post-mortem lividity of dependent portions. On right thigh over head of femur is found a large, deep bed-sore, fully 8 cm. in diameter. There is also a smaller, more superficial one over the left side in corresponding position.

"*Abdominal cavity* normal.

"*Pleural cavity* normal.

"*Pericardial cavity* normal.

"*Heart.* Not removed or opened.

"*Lungs.* Incision *in situ* shows considerable congestion and œdema, but otherwise negative.

"*Spleen.* Normal except for slight congestion.

"*Gastro-intestinal Tract.* Not opened.

"*Liver.* Surface everywhere smooth and glistening. In size the organ is normal. On section there is found to be slight congestion of bloodvessels, but surface markings are everywhere distinct. Consistency normal. There is absolutely nothing indicating a syphilitic lesion. Gall-bladder negative.

"*Kidneys* of normal size, capsule strips easily, surface markings fairly distinct. There is slight congestion.

"The above examination of organs was done very hurriedly and necessarily somewhat incompletely because of lack of time; the examination of the brain and cord having received the most careful attention.

"*Spinal Canal.* On removal of vertebral arches from the upper cervical region down to the cauda, the external surface of the dura mater covering the cord is found to show moderate congestion of the bloodvessels. There is also a slight deposition of fibrin over this surface in the dorsal region which is found blood-stained, probably from hemorrhage in removal of the cord.

"After the removal of the cord from the spinal canal and removal of dura mater, the following condition is found: There is no evidence of inflammation of the pia-arachnoid coat, although there is moderate congestion of the bloodvessels. In the dorsal region, however, beginning about 14 cm. (5½ inches) from the lower extremity of the cord and extending upward for a distance of 5 cm. (2 inches), a distinct fusiform swelling of the cord is seen. This swelling at the point of greatest thickness measures 15 mm. (⅝ inch), as compared with the adjacent cord, which measures 12 mm. (½ inch).

"The consistency of the swelling is firm, being slightly more so than that of the adjacent uninvolved cord. Meninges over this area show nothing remarkable. One centimetre (⅜ inch) above the upper border of this swelling there is another small nodule not more than 1 cm. (⅜ inch) in length. In this case as in the lower swelling the enlargement is symmetrical, but not quite so distinct; the diameter being not more than 14 mm. (½ inch). Section through these enlarged portions

of the cord reveals an apparent new-growth. All the surface markings normally found have been obliterated, and instead is seen a uniformly gray, more or less translucent tissue of rather firm consistency. There is no dilatation of the central canal. There is no evidence of hemorrhage in the involved area.

"*Brain.* There is marked uniform congestion of the bloodvessels of the pia-arachnoid coat. No evidence of inflammatory exudate. No thickening of meninges. No œdema. After removal of brain from skull there is found lying over the superior surface of the left cerebellar lobe a diffuse, irregularly lobulated tumor of fairly firm consistency. It extends practically from the median line separating the right from the left cerebellar lobe, over the superior surface of the left cerebellar lobe to the extreme left border, and from a point corresponding to the anterior border of the pons to within 1 cm. (⅜ inch) of the posterior border of the cerebellum. The tumor is roughly divided into three large lobes of irregular shape, with smaller lobes subdividing them. The borders are for the most part abrupt and in places give the impression of pedunculation, but elsewhere they seem to become lost gradually into the surrounding pia and brain tissue. There are no congested bloodvessels over the surface. Tumor mass is 2 cm. (¾ inch) above the general cerebellar surface. Section into tumor at one point shows the growth to extend fully 3 cm. (1⅛ inches) deep. In color it is gray and corresponds in general appearance to the masses found in the cord. There is no evidence of softening at the point incised. No hemorrhage. The inferior surface of the left occipital lobe shows slight flattening of convolutions corresponding to the situation of the underlying cerebellar tumor, and it is probable that the entire inferior surface of the left occipital lobe was subjected to direct pressure. As far as can be ascertained without considerable dissection, there is no connection of the cerebellar new-growth with any portion of the brain aside from the cerebellum.

"*Cerebellar Growth.* Sections from different portions show practically the same condition. The growth is found to consist of small, spindle-shaped cells of the embryonic, connective tissue type. These cells compose the bulk of the tumor; they are closely packed together and are found to follow no order in distribution and arrangement. In places there is a very delicate connective tissue stroma lying between the cells. This stroma when present in sufficient amount tends to form small alveoli in which the tumor cells lie. The bloodvessels are fairly numerous throughout the sections examined, and are seen to have very thin walls. In many places nothing but endothelium exists between the lumen and tumor cells. The underlying brain tissue is found to be infiltrated with tumor cells in varying degree. No evidence of degeneration can be discovered in any part of the tumor.

"*New-growths of the Spinal Cord.* They resemble in all respects the new-growth in the cerebellum. Sections from the larger growth referred to in the gross description reveal complete obliteration of the entire cord. The small, spindle-shaped tumor cells form one diffuse mass occupying the entire transverse section of the spinal cord. The pia-arachnoid coat is also involved. It is considerably thickened and shows everywhere in varying degree infiltration with tumor cells. This is especially well marked about the congested bloodvessels.

"In the centre of the growth occupying the cord are discovered

several irregularly shaped areas of small size which take up the eosin stain more markedly. Examination with high power shows few nuclei to be present and those which are do not stain well with hæmatoxylin. There is considerable finely granular detritus which stains light pink and a few compound granular corpuscles. No nerve fibres can be made out. Sections from the smaller of the two nodules referred to in gross description show practically the same condition as seen in the large one, but the process has not advanced so far. Although there has been complete replacement of the spinal cord by the new-growth, the pia-arachnoid coat is not invaded to such a marked degree. Throughout the pia there is considerable œdema, but very slight cellular exudate from the vessels. That which exists is confined entirely to the zone immediately about the vessels, which is also the seat of most marked growth of tumor cells. No areas of degeneration are to be found."

The examination of the sections from this case was not completed by Dr. Hendrickson, but further study has been made by me.

FIG. 1.

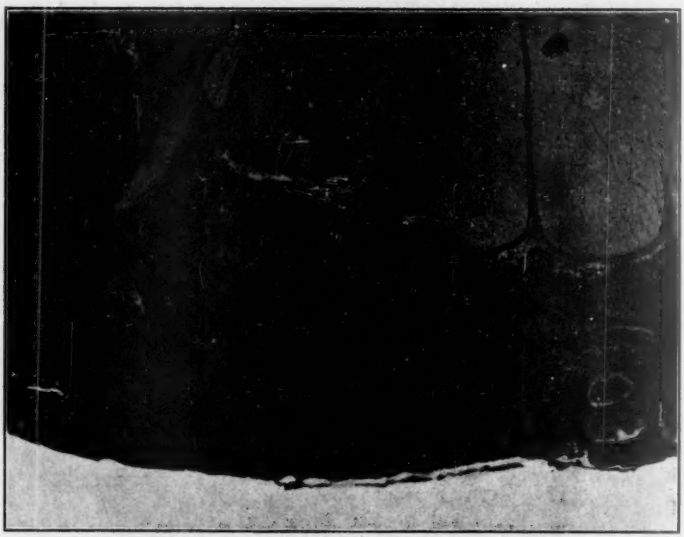
Photograph of a section from the thoracic region of the spinal cord in Case I., showing an intramedullary tumor surrounded by the partially degenerated spinal cord. The connection of the tumor with the pia is shown by the sarcoma cells within the pia of the anterior fissure, extending from the intramedullary tumor to the pia surrounding the cord.

Numerous small tumors are scattered throughout the pia of the spinal cord, especially in the posterior portion. The origin of one of the large intramedullary tumors of the cord can be traced distinctly to the pia. Cells are seen in great numbers extending through the pia of the anterior fissure into the substance of the cord, where they have proliferated to form the tumor, which is surrounded by a narrow band

of partly degenerated cord substance. (Fig. 1.) In the greater part of the spinal cord the tumors of the pia do not infiltrate from the pia into the cord substance, but here and there a beginning infiltration may be seen. (Fig. 2.)

The pia of the anterior fissure is infiltrated with tumor cells at portions where no tumor is found within the cord. The nerve cells of the anterior horns in the lower cervical and lumbar regions stain well by thionin, and appear to be in a very good state of preservation. The anterior and posterior nerve roots, though surrounded by tumor masses, appear to be in an excellent condition.

FIG. 2.



Photograph of a section from the spinal cord in Case I., showing the infiltration of the pia with sarcoma cells. In most places the sarcomatous tissue is sharply defined by the spinal cord, but at one side of the photograph the invasion of the spinal cord by the tumor cells is shown.

Sections from about the eighth cervical segment, and, therefore, above the intramedullary tumors, stained by the Weigert hæmatoxylin method show that the spinal cord was little degenerated. Scattered through these sections are small, faintly stained areas in which the nerve fibres have disappeared. The absence of pronounced ascending degeneration can be explained by the softness of the intramedullary tumors and the absence of compression and destruction of many axis cylinders passing through the tumor, and by the absence of degeneration in the posterior roots. The bloodvessels of the pia and of the spinal cord are not diseased.

Sections from the lumbar region stain well by the Weigert hæmatoxylin method, and do not show distinct degeneration.

The Marchi method could not be employed.

CASE II.—The patient, a man aged forty-two years, was admitted to the University Hospital, into the service of Dr. C. K. Mills, December, 1902, and later came under my care when Dr. Mills went off duty.

He was married and had five children. He had been a glass-worker and glass-blower all his life. His father had died at the age of sixty years; his mother at the age of sixty-two years, of Bright's disease. Two sisters are living and well.

He had never had earache or any discharge from the ears. About one year ago he began to complain of failure of eyesight. At that time he was able to work. A short time after his eyesight began to fail he began to have pain in the left occipital region extending to the left temporal region. The pain is now constant but much worse at night, especially late in the night or toward morning. He cannot lie on the left side because the pain in the head is increased by this position.

He sleeps better in a chair than in bed. For about six months he has had difficulty in walking; he becomes dizzy and falls. He has had two attacks of unconsciousness, one during the past summer, and one two weeks ago, lasting several hours. He did not have convulsions in either attack. At times he has attacks of vomiting without any apparent cause. He has a voracious appetite, and eructation of gas occurs after eating. He has also shortness of breath, some failure of memory, and at times homicidal impulses, especially when suffering intense pain.

The report on his aural condition given by Dr. B. A. Randall, December 29, 1902, is as follows: "He has a labyrinthine involvement; on right side apparently total, with marked deafness on the left side."

The report of the ocular examination made by Dr. E. A. Shumway, December 30, 1902, is as follows: "Optic neuritis (choked disk), O. D. Optic atrophy, O. S. Paralysis of external rectus in each eye. Convergent strabismus (paralytic). V.—R. E., 6/12 V.; L. E., fingers six inches. *Pupils:* O. S. larger than O. D., rather dilated. Both pupils respond to light. Diplopia impossible to elicit on account of poor vision in O. S. External rectus paralyzed on each side; eyes will not move beyond median line. Eye-grounds—Right: choked disk; difference of level of 4 D. ($1\frac{1}{3}$ mm.) between nerve head and retina; surrounding retina swollen, opaque; veins tortuous, dipping in and out of swollen retina. Left: atrophy of nerve, veins slightly tortuous, edges of nerve clean cut, no heaping up of connective tissue, arteries reduced in calibre."

An examination made by me December 31, 1902, gave the following results: The man is very deaf, but his intelligence is good and he replies promptly to signs. The facialis is not involved on either side. The sensory and motor portions of the trigeminus are not involved on either side. He contracts both masseters equally and well, and moves the lower jaw freely from side to side. He has no disturbance of sensation on either side of his face, either for pain or touch.

The tongue is protruded in the median line, but has a slight tendency to deviate to the left.

When the eyeballs are at rest they are in convergent strabismus, the right eye being turned more inward than the left. (Fig. 3.) When he attempts to move his eyeballs from side to side, neither eyeball passes

beyond the median line outward. He has no nystagmus, even in attempting to look to one side or the other.

He complains of soreness over the left supra-orbital and infra-orbital foramina, but has no tenderness over the left mental foramen, and no tenderness on pressure over the exit points on the face of the right trigeminus. The oculomotor nerve is not affected on either side.

The grasp of the hands is good on each side, and there is no impairment of the voluntary movements of the upper limbs. A slight tremor is noticed in each hand as he touches the first finger to the nose; this seems to be a form of ataxia. Biceps tendon, triceps tendon, and wrist reflexes are prompt on each side, and very little if at all exaggerated. The voluntary power in the lower limbs is normal. Gait is slightly ataxic with the eyes open, and the ataxia is a little increased when the

FIG. 3.

Photograph of the man described in Case II., showing paralysis of each external rectus, greater on the right side.

eyes are closed. The erect station is good with the eyes open or closed. The patellar reflex is a little exaggerated on each side. He has a slight tendency to ankle clonus on the right side, but not on the left. The Babinski reflex is not present on either side. The Achilles tendon reflex is a little prompter than normal on each side. Hemiasynergy cannot be obtained.

An examination made by Dr. Mills and myself January 1, 1903, gave the following results: The man says he can hear a bell when it is struck, he hears it in the left ear but not in the right, and he hears the voice but does not understand words, and has not been able to hear words during the past two weeks.

Sugar was not tasted at first on the left side of the tongue after thorough rubbing, but was tasted when it was rubbed over the entire

tongue. Salt was tasted promptly on the left side of the tongue. The sense of smell was preserved.

Percussion of the head just behind the left ear causes pain, but there is no tenderness on percussion of the left side. The man says that he has no pain in the right side of his head, but on the left side the pain begins in the back of the head and extends to the frontal region. The chief area of tenderness is just behind the left mastoid, and he flinches when pressure is made at this spot. He has less tenderness over the mastoid process.

The diagnosis of cerebellar tumor, probably on the left side, was made.

My examination on March 4, 1903, gave the following results: In walking he staggers slightly and his tendency is to go toward the left. Vertigo comes in attacks, and at these times he has great difficulty in walking because of his staggering gait. He has been seen in these attacks by the head nurse. The attacks of vertigo are of short duration, not over a few minutes. He was seen in an attack also by the resident physician, Dr. Hunter. The patient was sitting on the edge of the bed; his head fell forward on his chest, the trunk bent forward, the upper limbs shook, and the body lunged toward the left and slightly forward; this was followed by partial recovery and then another lunge. The attack lasted about one minute and a half.

The man's condition has not changed much since last December. The patellar reflex on each side is prompt and not much exaggerated. Ankle clonus and Babinski's reflex are not obtained, the movement of the toes being flexion from irritation of the sole of the foot. The Achilles jerk is prompter than normal on each side. The resistance to passive movements in the upper and lower limbs is normal on each side. The grasp of the hands is good. Tendon reflexes of the upper limbs (triceps tendon, biceps tendon, and wrist reflexes) are about normal. There is no involvement of the sensory or motor portions of the trigeminus or of the facialis on either side. Pain is promptly perceived in the face on each side. The corner of the mouth is well drawn up and the forehead well wrinkled on each side. The tongue is protruded straight. Nystagmus is not distinct, but when the patient moves his left eyeball far to the right a little inco-ordination is observed in this eyeball. Nystagmus probably is prevented by the paralysis of each external rectus.

The operation for removal of a cerebellar tumor was done by Dr. C. H. Frazier, March 6, 1903. The man was restless, delirious, and violent after the operation, and he never regained consciousness fully. The pulse rate was high, 140 at times. He died March 9, 1903.

The necropsy was made by Dr. Richard M. Pearce, who very kindly placed the material in my hands. Permission was obtained only for the removal of the brain and spinal cord.

A deep incision, extending about half-way into the left cerebellar hemisphere, with softened brain tissue about it, is the result of the operation.

A tumor 2 cm. ($\frac{3}{4}$ inch) in anteroposterior diameter and 2.5 cm. (1 inch) in lateral diameter was found in the angle made by the pons at its junction with the left cerebellar hemisphere. (Fig. 4.) The facialis and acusticus on this side were compressed by the tumor, and a smaller tumor was found enveloping these nerves and lying posterior

to the larger tumor. A tumor about 1 cm. (⅜ inch) in each diameter was found in the angle made by the union of the pons with the right lobe of the cerebellum, but it did not implicate the right facialis and acusticus. The left trigeminus at its entrance into the pons was enveloped in a small tumor mass.

The medulla oblongata was much enlarged, and the posterior portion of it fluctuated as though a cyst were contained within it, but this fluctuation was caused by a soft tumor filling the posterior half of the fourth ventricle and infiltrating slightly the dorsal part of the medulla oblongata. Although the circulation of cerebrospinal fluid within the fourth ventricle must have been disturbed by this growth, the fourth ventricle was not enlarged.

The right facialis and acusticus were embedded in a small, soft tumor at their entrance into the internal auditory meatus, and the right glossopharyngeus, vagus, and accessorius were embedded in a similar mass at their entrance into the jugular foramen.

FIG. 4.

Photograph of the cerebellum, pons, and medulla oblongata from Case II. A tumor is shown in the left cerebello-pontile angle, and the medulla oblongata is very much enlarged on account of a tumor on the floor of the fourth ventricle.

The pituitary body, optic chiasm, and each Gasserian ganglion were embedded in soft tumor masses. Each Gasserian ganglion was about twice the usual size on account of tumor formation about and within it.

A small papillomatous mass was found protruding from the dura over the front part of the second left frontal convolution, and when the dura was removed this mass was found to be adherent to the pia and to have penetrated the dura.

The left olfactory bulb was embedded in a small tumor.

Small tumors, some the size of a pin's head and some larger, were found scattered throughout the spinal pia and enclosing many of the spinal roots, especially in the cauda equina. (Fig. 5.) They were more numerous on the posterior aspect of the spinal cord. Some of these tumors in the cauda equina had a papillomatous appearance.

Several of the cranial nerves implicated by the tumors were cut in microscopic sections and examined.

Transverse sections of the left opticus showed that the sheath of the nerve was infiltrated by tumor cells, but nowhere within the nerve

were distinct tumor masses found, although there were many cells resembling those of the tumors scattered between the nerve fibres. Weigert hæmatoxylin sections of the nerve showed that much degeneration of nerve fibres had occurred.

Sections of the left Gasserian ganglion showed that the tumor had infiltrated between the nerve bundles and nerve cells of the ganglion. Some of the nerve cells were partially degenerated, having a shrivelled appearance, and the nuclei were eccentric. Some of the nerve cells stained very faintly and contained few chromophilic elements.

FIG. 5.

Photograph of the conus medullaris and cauda equina from Case II. Numerous small tumors are found growing about the nerve roots.

Sections of the right Gasserian ganglion stained by the Marchi method showed that the nerve fibres of the ganglion were in a moderate degree of recent degeneration.

The right abducens contained numerous areas free of nerve fibres, and yet by the Weigert hæmatoxylin stain the nerve appeared to be in a fair state of preservation. Degeneration was not found within the nerve by the Marchi method.

The left facialis was partly degenerated, as shown by the Weigert hæmatoxylin stain, and very intensely degenerated, as shown by the Marchi method. It is, therefore, remarkable that facial paresis did not exist.

The left acusticus was also much degenerated, as shown by the Weigert hæmatoxylin stain.

The right accessorius appeared to be normal by the acid-fuchsin and Weigert hæmatoxylin stains, but was shown to be distinctly degenerated by the Marchi method.

The tumor on the floor of the fourth ventricle infiltrated the dorsal part of the medulla oblongata, but did not invade the nuclei of the hypoglossal nerves.

Although numerous small tumors were present in the pia of the spinal cord, at no place were they found infiltrating the cord. They surrounded many of the nerve roots, and yet caused very little degeneration of these roots. Secondary degeneration, either ascending or descending, was not found in the spinal cord by the Weigert hæmatoxylin or acid-fuchsin stains, but very slight degeneration of the posterior columns, both in the cervical and lumbar regions, was detected by the Marchi method. Posterior root fibres could be seen passing through the small tumors of the pia, and yet staining well by the

FIG. 6.

Photograph of a section from the spinal cord in Case II., showing the infiltration of the pia with tumor cells. These cells have not invaded the spinal cord, and the nerve roots surrounded by the tumor mass appear normal. The columnar cells are seen in places.

Weigert hæmatoxylin or acid-fuchsin methods. A spinal nerve root embedded in a small tumor was found to be normal with the exception of a few slightly swollen axis cylinders. The bloodvessels of the pia and of the spinal cord were normal.

Many of the nerve cells in the anterior horns of the lumbar region stained by the thionin were much vacuolated, otherwise they were normal.

The resistance of the nervous tissue proper to tumor infiltration throughout the brain and cord is remarkable, and little degeneration has been caused by the numerous tumors, because they were soft and produced little pressure.

The tumors everywhere have much the same structure. They consist of round or somewhat elongated cells, and even by Mallory's

neuroglia stain contain very little intercellular tissue. In some places the cells are distinctly columnar and resemble those of the ependyma. (Fig. 6.) The columnar cells are arranged in long rows and have a large nucleus situated at one end of the cell. These rows of cells are found especially about the bloodvessels, but also where there are no bloodvessels. There is unquestionably a close resemblance between these cells and those of the ependyma, and the temptation, therefore, is to call the tumor an ependymoma, but it seems remarkable that an ependymoma, being a form of glioma, should give extensive metastasis to the pia of the spinal cord, which is of mesodermic origin. This, and the absence of glia fibres between the cells, and the distinct tendency to the formation of rows of cells about the bloodvessels, seem to justify the classification of the tumors under the sarcomata, possibly endotheliomata or peritheliomata, and yet the close resemblance of some of the cells to those of the ependyma may permit us to regard the growth as a mixed one, and as being partly a sarcoma and partly an ependymoma. The ependyma is known to proliferate in cases of syringomyelia when the cavity extends to the central canal, and to cover in part the wall of the cavity. It is not unreasonable to suppose that a sarcoma growing from the pia may extend to the fourth ventricle, and by irritation of the ependyma lead to its proliferation, and cause in this way a mixed tumor. This view is supported by another case in which a very similar tumor was found growing over the pons and implicating the posterior part of the fourth ventricle. A smaller tumor, probably metastatic, was found in the pia of the spinal cord, and the rows of ependyma-like cells were not so evident in this, although they were very striking in the large tumor. The tumors in Case II. have a resemblance to those described by Rosenthal,[1] Fraenkel, and Benda[2] as neuro-epitheliomota, and yet in some respects they are very different.

Case III.—J. C., aged thirty-nine years, a laborer, Italian, was admitted to the Philadelphia Hospital, in my service, June 28, 1902. He had paralysis of both lower limbs. His history so far as it could be obtained was as follows:

About three weeks before admission to the hospital he had had pain in the lower thoracic region and sensation of pressure about the waist, probably a girdle sensation. The pain had persisted in the abdomen and back. The left lower limb became weak, and soon the right lower limb was affected.

Notes made July 20th recorded that for three weeks he had had incontinence of urine and feces. He had not had pain in the lower limbs. The lower limbs were almost completely paralyzed. He was able on great effort to make slight movement of the right lower limb, chiefly with the thigh muscles. The limbs, except the soles of the feet, were not atrophied. The feet were œdematous and pitted slightly on pressure. The legs and thighs were not œdematous. The lower limbs were not contractured and were flaccid, especially the right. The patellar reflex was present on each side, but was diminished; the leg and foot on each side were very slightly moved voluntarily, but contractions were seen in the quadriceps muscles. The patellar reflex on the left side was a little prompter than on the right. Ankle clonus

[1] Ziegler's Beitrige, xxiii.

[2] Deutsche med. Wochenschrift, 1898, xxiv. p. 457.

and Achilles reflex were not obtained on either side, and the loss of these reflexes was not caused by rigidity of the muscles. Talipes equinovarus was present on each side, although the varus position was not pronounced. Babinski's reflex was not obtained on either side, and the toes did not move distinctly in either direction. The soles of the feet were scaly. Sensation for touch and pain was lost in the lower limbs and over the lower part of the trunk, both back and front, to a line passing about two inches below the nipples. A bed-sore was over the left buttock. The upper limbs and head were not affected. The right pupil, however, was much larger than the left. The spinal column was not deformed.

A diagnosis was made of transverse lesion of the upper part of the thoracic cord, causing complete or nearly complete destruction of the cord at this part.

FIG. 7.

Photograph of a section from the thoracic region of the spinal cord in Case III., showing the intramedullary sarcoma partially surrounded by the degenerated spinal cord.

On August 9th the man's condition was as follows: He had still incontinence of urine and feces, and had had this since his admission to the hospital. He was in semi-stupor. The face appeared emaciated. The upper limbs were well developed, but not muscular. The only voluntary movements possible in the lower limbs were in the flexor muscles of the thighs. Babinski's reflex was distinct on each side at this examination, but ankle clonus, Achilles tendon reflex, and patellar reflex were not obtained on either side. The plantar muscles of the feet were much wasted. The man died September 9, 1902.

After the spinal dura was opened an elongated swelling was found in the spinal cord just above the midthoracic region. This enlarge-

ment was about one inch in length. When a transverse cut was made through the middle of the swelling a tumor embedded within the substance of the spinal cord was found. No tumor could be detected elsewhere in the spinal cord, brain, or their membranes. Only the brain and cord were examined. The material was hardened in Orth's fluid.

A transverse microscopic section through the middle of the tumor shows that it is composed of spindle cells; it is, therefore, a large spindle-cell sarcoma, the cells being considerably larger than those within the tumor of the first case reported in this paper. The tumor fills the centre of the spinal cord and has a narrow band of cord substance about it, although on one side it extends to and invades the pia. (Fig. 7.) The cord substance at the periphery of the tumor is very much degenerated and stains poorly with the Weigert hæmatoxylin. The bloodvessels everywhere in the section are much congested. Round-cell infiltration is present about some of the bloodvessels of the pia, and infiltration of tumor cells is found in some of the nerve roots. Some of the nerve roots are degenerated.

Sections taken just below the tumor show the changes commonly seen in compression myelitis, viz., loss of nerve fibres, proliferation of neuroglia, some swelling of axis cylinders, and small, recent hemorrhages. The spindle cells of the tumor are not present at this level, either within the cord or pia. Sections stain well by the Weigert hæmatoxylin method, but contain numerous areas of necrosis. It is impossible to trace anywhere an origin of the tumor from the pia.

The nerve cells of the anterior horns in the lumbar region appear to be normal. Much degeneration is revealed by the Marchi method in the direct and crossed pyramidal tracts of the lumbar region. Flechsig's oval field in the posterior columns is partly degenerated. Sections stained by the Weigert hæmatoxylin method are a little pale; otherwise they appear to be normal.

Sections taken above and near the tumor stain well by the Weigert hæmatoxylin method, but the posterior columns, especially those of Goll, are partially degenerated. The anterior and lateral columns appear by this stain to be normal. Sections taken a little lower and at the upper border of the tumor reveal great degeneration, numerous necrotic areas, and much recent hemorrhage. Except at the level of the tumor sarcoma cells are not found in the pia.

Sections from the eighth cervical segment stained by the Weigert hæmatoxylin appear to be normal, except that the columns of Goll are much degenerated. By the Marchi method great degeneration is seen in the columns of Goll at this level, slight degeneration in the columns of Burdach, and very considerable degeneration in the direct cerebellar and Gowers' tracts and along the periphery of the antero-lateral columns. The nerve cells of the anterior horns at this level are deeply pigmented, but otherwise they appear to be very little altered.

A brief summary of the cases is as follows:

Case I.—The patient, a woman, had been well until one year after marriage. Three days after the birth of her child she had severe headache in the occipital region. After the headache ceased she was dizzy and had diplopia from external ocular muscle palsy. The lower limbs

gradually became stiff and weak, until finally they became completely paralyzed, and control over bladder and rectum was lost. Examination of the eyes showed paralysis of both external recti muscles, with paralysis of one of the oblique muscles, but the patient's condition made it impossible to determine which one. There was marked swelling of the optic disks, with hazy outlines, and the vessels were tortuous. Five months after the birth of her child her head was slightly retracted and the neck was a little stiff, and she moved the head from side to side with difficulty. The headache was still severe. The upper limbs were moved voluntarily and freely, and sensation in the upper limbs was normal. The lower limbs were completely paralyzed, and the patellar reflex was much diminished on each side. A slight ankle clonus was present on the right side, and Babinski's reflex was present on each side. All forms of sensation were lost in the lower limbs.

Two intramedullary sarcomata were found in the thoracic region of the spinal cord, and there were numerous small sarcomata in the spinal pia; a large sarcoma also was found in the left cerebellar lobe.

Case II.—The patient was a man, aged forty-two years. About a year before admission he began to have failure of eyesight and constant pain in the left occipital region. Six months later he began to have difficulty in walking and would get dizzy and fall. He had two attacks of unconsciousness, but without convulsions. He had also attacks of vomiting Dr. B. A. Randall found labyrinthine involvement, apparently total on the right side, and on the left side the deafness was intense. Dr. E. A. Shumway reported that the man had choked disk in the right eye and optic atrophy in the left. Both external recti muscles were paralyzed. The man had a little tenderness over the left supra-orbital and infra-orbital foramina. He had occasionally attacks of vertigo.

An attempt was made by Dr. C. H. Frazier to remove a cerebellar tumor which was believed to be present, and probably on the left side, but was unsuccessful. The operation in this case will be described by Dr. Frazier. A tumor was found in each cerebello-pontile angle, but the larger one was on the left side. Tumors were found about the Gasserian ganglia, pituitary body, floor of the fourth ventricle, right internal auditory meatus, and right jugular foramen, and numerous small tumors were found in the pia of the spinal cord.

Case III.—The patient was a man who had had pain in the lower thoracic region and girdle sensation about three weeks before admission to the hospital. The pain persisted. The left lower limb became weak, and this was followed by weakness of the right lower limb. He had incontinence of urine and feces. He did not have pain in the lower limbs, but the lower limbs became almost completely paralyzed and flaccid, and sensation was lost in these parts and in the trunk

as high as a line passing about two inches below the nipples. The tendon reflexes of the lower limbs were absent, but Babinski's reflex was obtained on each side. A primary sarcoma was found within the spinal cord just above the midthoracic region.

Sarcomatosis of brain and cord and of the pia covering them may cause great difficulty in diagnosis, and in some cases a correct diagnosis may be impossible, as in the second of the cases here reported. Extensive alteration may cause few symptoms, and this has been remarked upon by a number of authors. Schlesinger[1] speaks of it in his monograph on spinal tumors, and it was true of Hippel's[2] case, in which the sarcoma cells pushed apart the elements of the nervous tissue. In A. Westphal's[3] case, also, the extensive sarcomatosis was remarkable in that it caused comparatively few symptoms. The importance of this knowledge, when a question of operation is under consideration, is great, because, if evidence of sarcomatosis is found, the case is an inoperable one. The soft tumor masses grow in the pia about the spinal cord and about the nerve roots, especially those of the cauda equina, and about the cranial nerves, and cause few clinical signs of their presence, because they produce little or no compression or destruction of nervous tissue. In a case like Case II. of this paper, the pia of the spinal cord and brain and the dura at the base of the brain may be extensively infiltrated with sarcoma cells, the Gasserian ganglia and cranial nerves may be embedded in tumor masses, and yet few signs of disturbed function be manifest.

Westphal especially emphasized that sarcomatosis of the membranes has little tendency to invade the spinal cord, whereas in tuberculosis and syphilis the invasion of the nervous tissues is common. He also mentions that in all the recorded cases the posterior part of the pia was the part most affected. This part was also most affected in Case I. and Case II. Sarcomatosis of the membranes of the cord and brain is, according to him, a rapidly fatal disease.

C. Busch[4] remarks that his case of sarcomatosis of the pia, as well as the cases of Schataloff and Nikiforoff afford proof that sarcoma of the pia has very little tendency to invade the spinal cord and extramedullary roots, even when it surrounds these structures. Case II. of this paper is further evidence of the correctness of this opinion, but Nonne[5] says that sarcomatosis may invade the nervous tissues, and he gives as examples the cases of Bilfenfeld and Benda. He quotes Bruns as saying that the process has very little tendency to implicate

[1] Beiträge zur Klinik der Rückenmarks- und Wirbeltumoren.
[2] Deutsche Zeitschrift f. Nervenheilkunde, vol. ii. p. 388.
[3] Archiv f. Psychiatrie, 1894, vol. xxvi. p. 770.
[4] Deutsche Zeitschrift f. Nervenheilkunde, vol. ix. p. 114.
[5] Ibid., vol. xxi., Nos. 5 and 6, p. 369; Archiv f. Psychiatrie, vol. xxxiii. p. 393.

the nervous tissues, whereas Schlesinger remarks that a direct invasion of the spinal cord by the sarcoma cells is frequent. The resistance of the extraspinal roots to the process has been observed by A. Westphal, Schlesinger, Nonne, Benda, and others.

Case I. of this paper shows that occasionally sarcomatosis may extend into the substance of the spinal cord and brain, and both Case I. and Case II. afford evidence of the resistance of the nerve roots to infiltration by tumor cells.

It is partly because of this escape of the nervous tissue in many cases that a correct diagnosis of the extent of the process may be impossible.

The infiltration of the pia resembles that caused by syphilis or tuberculosis, for the process is, if we care to so call it, largely a sarcomatous meningitis. An incorrect diagnosis of syphilis is not unlikely. Nonne called attention to the resemblance of a case of his to one of acute cerebro-spinal syphilis, but no history of syphilis and no clinical signs of this disease could be discovered, mercury and iodide were without effect, and such severity of symptoms and rapidity of development he thought were very uncommon in syphilis. To the naked eye the brain and spinal cord in Nonne's case appeared to be normal, and yet the pia everywhere was infiltrated with cells which were believed to have had their origin from the endothelium lining the lymph spaces about the vessels—*i. e.*, the so-called perithelium. The infiltration was especially marked about the optic chiasm and over the pons, and was greater over the posterior portion of the cord than over the anterior. This method of selection is like that occurring in syphilis. The cells invaded the nervous tissue at different places, and this invasion was especially by means of the pial septa. In the cervical region infiltration into the cord was by way of the anterior fissure, as in the case of Mrs. K. (Case I.). A difference from syphilis was found in the absence of circumscribed tumors, so common in syphilis; in the absence of disease of the vessels, and in the uniformity of the infiltration of the pia. The latter without the formation of small tumors here and there was very remarkable, and it seems to be the only case of the kind. The symptoms at first indicated hysteria and later a diffuse process of brain and cord.

The possibility of syphilis in the case of Mrs. K. (Case I.) and also in Case II. was carefully considered. The symptoms in Case I. were strongly suggestive of syphilis, and they had developed a year after the patient's marriage; it was therefore possible that she had contracted syphilis from her husband. The process was evidently a diffuse one involving the brain and spinal cord. Syphilis could cause just such a symptom-complex, but the cerebral symptoms and complete paralysis of the lower limbs without impairment of function in the upper limbs was unlike a syphilitic symptom-complex. It is true that the thoracic

region is often the most diseased part when syphilis attacks the spinal cord, but it is not common even in such cases to find an entire absence of symptoms indicative of implication of the cervical region of the cord. If weakness does not develop, at least pain in the upper limbs is common when the syphilitic process invades the brain and spinal cord. This apparent escape of an intervening part of the central nervous system is suggestive of sarcomatosis, because the sarcoma cells may invade the spinal cord only at one place and cause disturbance of function in the portion of the body innervated from the cord below the diseased area, while the soft tumor masses of the pia above produce no symptoms.

In Case II. syphilis was regarded as possible and antisyphilitic treatment was employed without very brilliant results. The bilateral deafness, the paralysis of each external rectus, the choked disks, the headache and attacks of vertigo could be easily explained by syphilis, and yet the case was one in which tumor cells infiltrated between and about many nerve fibres, and even between the nerve cells of the Gasserian ganglia, and caused very few symptoms.

In F. Schlagenhaufer's[1] case both trigeminal nerves and the optic chiasm were embedded in tumor masses, but microscopically the tumor cells were found to implicate the pia alone about these nerves. A small-cell sarcoma in the cervical region was believed to be the primary growth, but sarcoma cells were found in the pia of the brain and of the spinal cord. Schlagenhaufer thought that the sarcoma of the cervical region had its starting point within the cord, and was not the result of extension of a growth into the cord from the pia. He believed that to Schlesinger's classification he could add another division, viz., primary sarcoma of the spinal cord with secondary sarcomatous infiltration of the pia.

Schlagenhaufer's case resembles Case I. of this paper in the involvement of the cord by a tumor, and yet the origin of one of the tumors in Case I. could be traced from the pia surrounding the cord through the pia of the anterior fissure. Schlagenhaufer's case resembles Case II. in the implication of cranial nerves by the tumor masses.

Inasmuch as the symptoms in Case I. began three days after the birth of a child, it seemed possible that septic infection had occurred, and that the patient was suffering from purulent meningitis. The duration of the symptoms for five months without any signs of disease of the cervical cord was rather against the diagnosis of purulent meningitis.

That sarcomatosis may give the signs of tuberculous meningitis is shown by a case in a child, aged four years, reported by Lereboullet.[2]

[1] Obersteiner's Arbeiten, vol. vii. p. 208.

[2] Abstract in Revue Neurologique, January 30, 1902, No. 2, p. 98.

In Cases I. and II. reported in this paper both abducent nerves were paralyzed, and yet in neither case was there any direct implication of these nerves or their nuclei by the tumor masses; paralysis of both external recti, therefore, does not prove that a tumor is situated on or in the pons.

Tenderness of the vertebral column was one of the first and most striking signs in A. Westphal's[1] case, and it has been observed in other cases, and is suggestive of meningitis, but it is not always present in sarcomatosis of the spinal pia. It was not present in Case II., but was present over a portion of the spinal column in Case I.

Syphilis has sometimes caused the clinical picture of disseminated sclerosis, and therefore it is not surprising that sarcomatosis should simulate the same disease, as in a case reported by Hippel.[2] The symptoms in this case were optic neuritis passing into atrophy, nystagmus, headache, vertigo, scanning speech, progressive dementia, vomiting, intention tremor, exaggerated tendon reflexes, ataxic gait, and pain in the extremities. The findings were: sarcoma of the right half of the cerebellum; multiple sarcomata of the cerebral and spinal dura, of the pia, choroid plexus, brain, and spinal cord; diffuse sclerosis of the central nervous system, and metastatic sarcomata of the skin.

In this case, as in Case II., a tumor was found at the internal auditory meatus, and in two cases reported by Soyka (cited by Hippel), which were probably cases of sarcomatosis, a tumor was found in each at the internal auditory meatus; so that this, like the pontile cerebellar angle, seems to be a favorite location for sarcoma.

In Case II. the age was more advanced than that at which sarcomatosis is most common. A. Westphal says that most persons with sarcomatosis of the central nervous system have been very young, and that two-thirds of the cases have occurred in childhood. In Case I. the age was twenty-one years.

Schlesinger, in his excellent monograph on spinal tumors, divides primary sarcoma of the nervous system into: 1. Solitary sarcoma. 2. Multiple sarcomatosis.

Tumors of the first group may occur: 1. In the substance of the spinal cord. 2. Primarily in the meninges or nerve roots and (*a*) remain limited to these parts, or (*b*) invade the spinal cord.

Multiple sarcomatosis may occur as: (*a*) Disease of the nervous substance and meninges. (*b*) Multiple sarcomatosis of the membranes without sarcoma of the cord or brain, when it is (1) in the form of multiple small tumors, or (2) as diffuse sarcomatous infiltration of the membranes.

[1] Loc. cit. [2] Loc. cit.

Schlesinger succeeded in finding in the literature (1898) 18 cases of primary sarcomatosis of the central nervous system with implication of the cord or its membranes, and to these he adds 2, making 20 cases in all. In 14 of these cases implication of the brain and cord, or of their membranes, occurred. In 9 of these 14 cases cerebellar tumor was found, and in 3 the medulla oblongata was affected. It appears, therefore, that when the brain or its membranes are implicated in sarcomatosis, usually the structures of the posterior cranial fossæ are affected, and that in about two-thirds of the cases a tumor of the cerebellum is found. The diffuse infiltration of the spinal membranes seems to be more extensive in the lower portion of the vertebral canal.

In a case of Bruns and in one of the Vienna Pathological Institute the sarcomatous process extended entirely across the cord. In other cases of sarcomatosis of the nervous system an isolated tumor has been found within the cord, separated by cord tissue from the sarcoma cells of the pia.

Disease of the walls of the bloodvessels, according to Schlesinger, has been observed in only a few cases of sarcomatosis. The vessels were normal in both Case I. and Case II.

Since the publication of Schlesinger's monograph a few more cases of sarcomatosis of the central nervous system have been reported.

The occurrence of a cerebellar tumor in so many of these cases is remarkable, and recently Henneberg and Koch[1] have mentioned that the angle made by the pons and the lobe of the cerebellum on either side is a common seat for tumors of the posterior fossæ. The symptoms seem to indicate that these tumors arise most frequently in the acusticus, but it is possible that the acusticus is the cranial nerve first compressed. These authors point out that the tumor is more frequently on the left side, in the proportion of 3 to 2. It was on the left side in both Case I. and Case II. They say that often neurofibromata cause no disturbance of function in the affected nerves; so that this absence of symptoms is as true of fibromata as of sarcomata.

One of the most recent papers on the so-called tumors of the acoustic nerve is by Fritz Hartmann.[2] He calls the location in which they commonly occur the recessus acusticocerebellaris, and he, as others have done, emphasizes that these tumors are loosely attached to the brain, and that their attachment may be easily ruptured. The tumors are usually in organic relation with the acusticus, more rarely with the facialis, and in some cases the acusticus entirely disappears within the tumor; consequently, deafness of central origin is one of the earliest symptoms, as it was in my Case II.

On account of the frequency of tumors in the cerebello-pontile angle,

[1] Archiv f. Psychiatrie, vol. xxxvi., No. 1, p. 251.
[2] Zeitschrift f. Heilkunde (interne Medicin), 1902, vol. xxiii. p. 391.

such as occurred in Case II., the important paper relative to operation on the posterior cranial fossa recently published by F. Krause may be referred to here.

He has shown by a successfully operated case that it is possible to divide the acusticus intracranially for the relief of severe tinnitus aurium. He entered the skull through the posterior fossa and divided the acusticus. The tinnitus was lessened, but the patient died from pneumonia five days after the operation.

In another case Krause[1] exposed at different operations both cerebellar hemispheres, and although a cerebellar tumor was not found, but internal hydrocephalus was, the condition of the patient was improved, and death did not occur until three years after the operation. The case showed that a cerebellar hemisphere might be incised to a depth of 2 or 3 cm. ($\frac{3}{4}$ to $1\frac{1}{4}$ inches) without causing serious symptoms.

Krause has also exposed both cerebellar hemispheres by one flap. The patient died six days after the operation, and was found to have internal hydrocephalus and deformity of the base of the skull. Although such an opening permitted a view of a large part of the cerebellum, it does not follow necessarily that it would have made possible the removal of a tumor situated in the cerebello-pontile angle without causing the death of the patient. A tumor so situated in a case of M. Allen Starr was removed, but the patient died a few days after the operation.

The primary sarcoma within the substance of the spinal cord in Case III. is a very uncommon form of tumor. It is primary, at least so far as the central nervous system is concerned.

Schlesinger[2] says that primary sarcoma of the cord without implication of the meninges, especially exclusive of the gliosarcoma and the myxogliosarcoma, is of very rare occurrence. He has been able to find only 13 cases. The tumor is usually sharply defined from the surrounding cord tissue, and sometimes encapsulated, which he thinks is important surgically, and yet I do not believe that an encapsulated tumor of the spinal cord can be removed with much benefit to the patient. In 8 cases the primary sarcoma was in the cervical region, in 1 in the midthoracic region, in 1 in the lower thoracic region, in 1 it extended throughout the entire thoracic and lumbar regions, in 1 it was in the lumbar region, and in 2 it extended throughout the length of the spinal cord. In 2 of the 13 cases cavities in the cord were found. In my case the sarcoma was in the thoracic region.

[1] Beiträge zur klin. Chirurgie, vol. xxxvii., No. 3, p. 728.
[2] Loc. cit.

HISTOLOGICAL CHARACTER AND DIAGNOSIS OF MALIGNANT NEOPLASMS OF THE DIGESTIVE ORGANS AND PERITONEUM.

By John C. Hemmeter, Ph.D., M.D.,
PROFESSOR IN THE MEDICAL DEPARTMENT, UNIVERSITY OF MARYLAND, AND DIRECTOR OF THE CLINICAL LABORATORY.

The progressive enlargement, not restricted by any obstacle, and the destructive or corroding advancement of malignant growths have given rise to the impression that cancer cells possess peculiarities of growth not found in other neoplasms. It has been suggested by Hansemann and others that the cells, through the proliferation of which malignant tumors arise, had undergone a change in their biological character. Many efforts have been made to obtain a clear conception of this change of quality, but they have not been successful. Hansemann speaks of an antecedent change in the biological character of the cells, which is claimed to be necessary for pathogenesis of malignant neoplasms, but he does not define this change. In other words, an unknown, indistinct hypothetic factor is introduced, with the hope of clearing up our understanding of the origin of malignant neoplasms. It is evident that nothing is gained by attributing the pathogenesis of such growths to a change in the cells when this change itself remains incomprehensible and undefined. Ribbert[1] believes that the physiological processes of growth are sufficient to serve as guiding lines in the development of neoplasms. Hansemann has attempted to make the stronger ability to grow comprehensible by assuming that the cells lose their characteristic peculiarities, a loss of their cellular individuality, whereby they are assumed to revert to a less differentiated stage, approaching again a condition more like the cells of the ovum. This reversion or change of cells he calls "anaplasia"—(to build backward) undifferentiation. Hansemann based his conception upon deviations of tumor cells from the character of the normal cells from which they originated. Frequently the neoplastic cells show a simpler morphological quality and structure, which he interprets as a lesser degree of differentiation. For example, pavement epithelium loses its tendency to become hard or horny-like epidermis, and in cancers may represent morphologically indifferent cell forms. But this simplification of the structure of tumor cells is frequently not found in cancers. For example, the pavement epithelium of the epidermic cancer may show extensive cornification in the lymph glands and secondary metastases, and is indistinguishable from the epithelium of the primary seat

[1] Pathol., s. 6, 10.

of origin. The cylindrical cell cărcinoma may form mucus in the secondary nodules. The osteosarcoma produces calcified bone substance in the metastases found in internal organs.

The anaplasia of Hansemann becomes less and less evident the nearer the neoplasm is traced to its beginning stages, that is, in those stages where it would count most as an etiological factor it is less evident. In order to be considered a cause it should precede the development of a neoplasm, not follow it. Ribbert considers the anaplastic changes as secondary, as a reversion. Hansemann holds that new cells, peculiar to themselves, arise by this process. This is denied by Ribbert, who interprets it as a reversion to a previous stage in development.

Hansemann[1] first used the word "anaplasia" in 1890, the prefix being *ava* (from *ava*, backward, and *πλάσσειν*, to build, building, "zurück," "backward"). He understands by it a condition in which the cells have lost, in part, their specificity, have become "entdifferenzirt," so that they have acquired the property of independent existence, have lost what he calls "altruismus." He conceives that this anaplasia is brought about by abnormal, especially unsymmetrical, mitoses, and that some of the *idioplasm*[2] is thereby lost, while idioplasm which had previously been in the background now comes to the front.

Hansemann does not consider the anaplasia in itself sufficient to produce malignant neoplasms, but emphasizes that a special factor, agent, or irritant is necessary in addition to cause the anaplastic cell to proliferate into a tumor. This agent has been supplied in the argument of those who hold the view of the parasitic origin of cancer. The efforts to demonstrate characteristic bacteria as causes have been in vain. The protozoa have been blamed as the cause of cancer, but most careful researches of many competent observers have resulted in the consensus of opinion that the curious enclosures thought to be protozoa are degeneration products of cells and nuclei enclosed by leucocytes and lymphocytes.

It is a characteristic of malignant tumors that cells of other types than the original are never involved in the growth—a pavement epithelial cancer never involves cylindrical epithelia. This necessi-

[1] Virchow's Archiv, Bd. cxix. p. 321.

[2] *Idioplasm.* In biology a term introduced by Nägeli for a special hereditary reproductive substance not contained in the body of the cell, but in the *chromosomes* of the nucleus controlling and determining the *actual* characters of the particular cell, and also those of all of its descendants. Each idioplasm is composed of several or many *ids*, which are capable of growth and multiplication by division; although much smaller in bulk than the rest of the living substance of the cell or body (*trophoplasm*), idioplasm is the active element in the process of formation, and determines the detailed construction of the trophoplasm, which is the passive element.—*Gould's Dictionary of Medicine.*

tates the assumption, if we adhere to the bacterial hypothesis, that every kind of malignant tumor must have a particular species of parasite as its cause. Our understanding of the pathogenesis of tumors is made difficult by trying to apply the bacterial theory in its explanation.

A schema which should indicate that a neoplasm which corresponds to a certain type is benign and another that looks different is malignant is a conception that becomes impossible with closer and careful study of their growths. We should never approach a tumor with the question, "Is it a carcinoma or a sarcoma?" But the diagnosis should be developed like that of the disease in the living subject. First, we must consider the anatomical properties; second, the topographical; third, the distribution of the various tissue types in the tumor, and, fourth, the properties and peculiarities of the cells. Then, if at all, the diagnosis follows naturally of itself.

If the question is to make a diagnosis from a fragment of an organ separated from the body, we must first ask ourselves the question, "Is this a real tumor or is it an inflammatory proliferation, a simple hyperplasia, or infective tumor?" Theoretically it would seem easy to distinguish the inflammatory tumor from neoplasms, and yet there are cases where considerable difficulties arise, and, indeed, a confusion may occur in two ways: first, when a real neoplasm is taken for an inflammatory proliferation; second, when the latter has the appearance of a genuine neoplasm. The first danger is most easily avoided by very careful and thorough observation. It often happens that the stroma of the tumors is developed so greatly or infiltrated with small cells to such a degree that the real parenchymatous islands are only discovered after a protracted search. This is on record as having occurred with the scirrhi, which for a long time were classed with the fibromas or inflammatory thickenings.

Hansemann gives an interesting description of sections made from excisions of the vaginal portion of the uterus. The preparations gave a strong impression of a granular proliferation strongly infiltrated with cells. Newly formed vessels were everywhere present, which were filled with leucocytes, and the whole picture was flooded with leucocytes to such an extent that at first nothing was seen but these. It was only after a more thorough examination that he found groups of cells rich in protoplasm which were in several places arranged like epithelia, and were very different from the swollen endothelia. Without doubt it was a cancer.

The cancerous portions are also easily overlooked in old indurated ulcers, and a long series of preparations must first be made before we can arrive at a certain diagnosis. It happens much more easily that inflammatory neoplasms are considered malignant tumors.

Both epithelia as well as connective tissue substances may form such great proliferations by means of inflammatory changes, which may show such a cellular composition that considerable practice and experience are necessary not to confound these with cancer and sarcoma.

In January, 1900, I came into possession of a specimen of intestinal tissue from a case of intussusception. At the tip of the invaginated part there was a circular swelling almost as thick as a finger, which might have been mistaken for a malignant tumor, and consequently as the cause of the invagination. The microscopic examination showed this not to be the fact. The thickening consisted of plentiful connective tissue with elastic fibres and fatty tissue. It was manifestly a case in which the intestinal wall and the serosa were thickened by inflammation.

Extreme difficulties arise when the endothelia have led to especial proliferation. It very often occurs in simple inflammation in various portions of the body that these cells increase to such a degree that the microscopic image shows a distinctly alveolar construction. This is found especially often in the pleura if a thick callosity is formed. But I have observed this alveolar structure in a benign neoplasm of the ileocæcal valve, and in one of the ileum. Whether at this place the mass of the cells take their origin from the epithelia of the pleura or intestine, or from the lymph-endothelia, has been the subject of much discussion. This point Hansemann does not consider as yet decided. They have, however, often been considered tumors.

In this connection two interesting cases are described by Hansemann. In both cases trauma had preceded. In one case the cheek had received a blow from a beam, in the other a rusty nail had penetrated the hand and remained there for some time. In the course of the succeeding weeks a small tumor developed in each case, and these tumors were very similar. They consisted of large spindle cells and round cells, rich in protoplasm, with tender intermediate substance, scanty stroma, and very abundant pigment. In the tumor of the cheek the pigment lay everywhere between the cells, and, therefore, arose from the bleeding caused by the blow. In the other case, however, the cells had taken up the pigment, and thus a complete similarity with a melanosarcoma arose. However, the pigment had one property which is not observed in melanosarcoma, it had a strong iron reaction, and, therefore, it was reasonable to assume that the pigment arose from the rust of the nail, and had been taken up by the cells. The simple extirpation of both tumors caused a permanent cure.

Proliferations of the connective tissue may not only have the appearance of real tumors, but inflammation may accompany the pro-

liferation of the epithelia. Friedländer has devoted a noteworthy treatise to these, and characterizes this growth of the epithelia as an atypical proliferation. They are found extremely frequently in the various chronic inflammatory processes of the epidermis, which also accompany proliferations of the connective tissue and the formation of granular tissues. The border of the epithelium grows deep down into the granular tissue on the edges of chronic cellulites of old ulcers, in lupus, etc., branches off in different ways, and finally forms lumps and nests of epithelial cells. They contain many mitoses and also often pathological and atypical forms. In many cases the similarity to cancers may thus become so great that a mistake may very easily be made. Hansemann[1] has seen cases in which a differentiation from real cancroids was no longer possible in the coarse structure of the proliferations and in which the clinical development demonstrated its nature. In other cases the diagnosis was made possible at once by the knowledge of the condition which this investigator designates as "anaplasia." Hansemann takes this opportunity to point out especially the similar state of affairs in the larynx and at the vaginal portion of the uterus. Proliferations of the epidermis are often found so extended in the larynx, especially in tuberculosis, more rarely in syphilis, that the perfect structure of cancroids is formed. If, in such cases, the entire larynx can be sectioned, tubercles will always be found somewhere, which will render the diagnosis possible. If, however, only an excised specimen is at hand during the examination, the diagnosis becomes extremely difficult, or even impossible. (At the vaginal portion of the uterus the so-called erosions may cause a similar confusion. These do not consist of wound surfaces, as has been heretofore assumed, but of islands of cylindrical epithelium which has forced itself from the neck of the uterus between the normal pavement epithelium of the vagina. Such conditions are found in small children, and are probably inherited.) Later on, if inflammation arises here, or if the vaginal portion of the uterus is injured during birth, the cylindrical epithelia sometimes grow downward like a hose and assume a striking similarity to destructive adenomas, into which they may really develop. On this account it is the more important to know that certain histological proliferations exist which bear the greatest similarity to cancers, and yet are not cancers. It is very probable that all these cases of cancroids said to have been cured by internal remedies are such atypical proliferations of the epithelia.

Tubercular Tumors. Infectious tumors of the stomach and intestines are occasionally confounded with sarcomas. As a rule, it is not difficult to differentiate an intestinal retroperitoneal or omental tuber-

[1] Mik. Diag. d. Bösartigen Geschwülste.

cular tumor from a sarcoma. Yet I have seen cases where such tuberculous masses acquired such an extent and showed such a structure that they are not unlike sarcomas. Virchow himself at one time classed tuberculous tumors of cattle (perlsucht) among the sarcomas, on account of their fibrous structure, their cellular development, and their frequently scanty caseation. Similar perlsucht-like knots, resembling bovine tuberculosis, sometimes occur in man in the peritoneum and the pleura.

Hansemann[1] described two pulpy retroperitoneal glands, as large as the fist, without tubercles, with a minimal caseation and few tubercle bacilli. The characteristic giant cells are probably seldom found wanting in these affections. Tubercle bacilli are also present, but often in such small numbers that they are not discovered under the microscope. The transmission, however, usually succeeds on guinea-pigs, which in such cases always succumb under the form of tuberculosis peculiar to them.

Actinomycosis of the stomach and intestines may present tumors at the operation or at autopsy which outwardly may simulate malignant neoplasms, but microscopic examination, as a rule, shows the characteristic structure (globules) of the fungus actinomyces.[2]

Typhoid Proliferations. If the neoplastic indurations of enteric fever (abdominal typhoid) should be encountered at operation or necropsy, they can be recognized without difficulty when fragments thereof are examined under the microscope. Only in cases of lymphoma of the liver might a possible confusion with malignant tumors arise.

The purely lymphatic character of the same characterize them well enough to distinguish them from sarcomas; they might possibly be confused with leukæmic or syphilitic proliferations. Sometimes melanosarcoma in the intestines forms similar ulcers of a strongly gummatous appearance, like typhoid in its early stages. If the amount of pigment contained in them is considerable, this alone suffices for a macroscopic differentiation. These melanotic proliferations also are not confined to the Peyer's patches and the follicles. Otherwise, however, the histological appearance of these sarcomas is so characteristic that they cannot easily be confused with the lymphatic proliferations of typhoid fever.

The gland tumors of the plague have recently been thoroughly investigated by Aoyama, and his investigations have been confirmed by Hansemann. The clinical progress of this disease probably rarely necessitates a differential diagnosis between these tumors and sarcomas. It is well known, however, that in all epidemic diseases, and especially at the close of an epidemic, rudimentary cases of plague occur which

[1] Loc. cit.

[2] Hemmeter. Diseases of the Intestines, vol. ii. p. 108.

may then lead to difficulties in the diagnosis. According to Aoyama and Hansemann, the buboes of the plague show a proliferation of all three component parts of the lymphatic glands—of the lymphocytes, of the endothelial and of the connective tissue lattice-shaped cells. At the same time great hyperæmia, accompanied with hemorrhages, exists at first. Later on necrosis occurs in the tissue, and also suppurative softening. Finally the glands may become fibrous, hardened knots. The bacteria are present in great numbers in the glands, and may easily be detected by means of aniline colors, but not by Gram's method. Generally we are confronted with tumors which are not unlike typhoid lymph glands, only of an excessive size, and which can easily be distinguished from malignant tumors by their histological structure.

In a report from the United States Treasury Department, entitled "Bubonic Plague," by Walter Wyman, Surgeon-General Marine Hospital Service, it is stated (page 36) that intestinal plague is very rare, and Dr. H. F. Müller, in his valuable monograph "Die Pest,"[1] states that the anatomical changes in the digestive tract are not important in this disease. The mesenteric and solitary glands may be swollen, but they never acquire the size of deserving the name of tumors. In the stomach there are superficial hemorrhages. Wilm[2] describes the solitary follicles as swollen and reaching the size of a bean. The Peyer's patches were hyperplastic but not ulcerated. It is evident from these quotations that even in countries in which the plague occurs epidemically it rarely effects striking changes in the digestive tract, and should they occur they are not likely to be confused with malignant neoplasms.

Leukæmia, Pseudoleukæmia, Syphilis. These diseases may present difficulties for the differential diagnosis from malignant neoplasms. They may all give rise to the formation of large tumors. Leukæmia and pseudoleukæmia do not differ from each other in the microscopic pictures of their tumors. In both the tumors of the lymphatic glands are composed of typical lymphatic cells. However, the follicles, the proliferating centres of the lymphatic glands, have disappeared. The cells are slightly larger than the normal lymphocytes and show numerous mitoses of the typical form of the lymphocytes. Also in the infiltrations of the tissues of the kidneys, spleen, liver, and testicles the typical lymphocytes are everywhere found. Growing over into neighboring tissues and spreading out beyond an organ by means of propagation, they are never observed, and the constituent parts of the infiltrated organs are not destroyed, but only pressed asunder mechanically. It is for this reason that a liver or a kidney

[1] Nothnagel's Spec. Path. u. Ther., Bd. v. p. 41. [2] Hygen. Rundschau, 1897, p. 222.

that has been completely infiltrated by syphilitic neoplasms, for instance, may yet function completely, and that neither uræmia nor jaundice usually appears as a result of the leukæmic proliferations pure and simple.

The ordinary syphilitic gumma of the gastro-intestinal canal (Fig. 1) can usually be distinguished without any difficulty from sarcomas, even

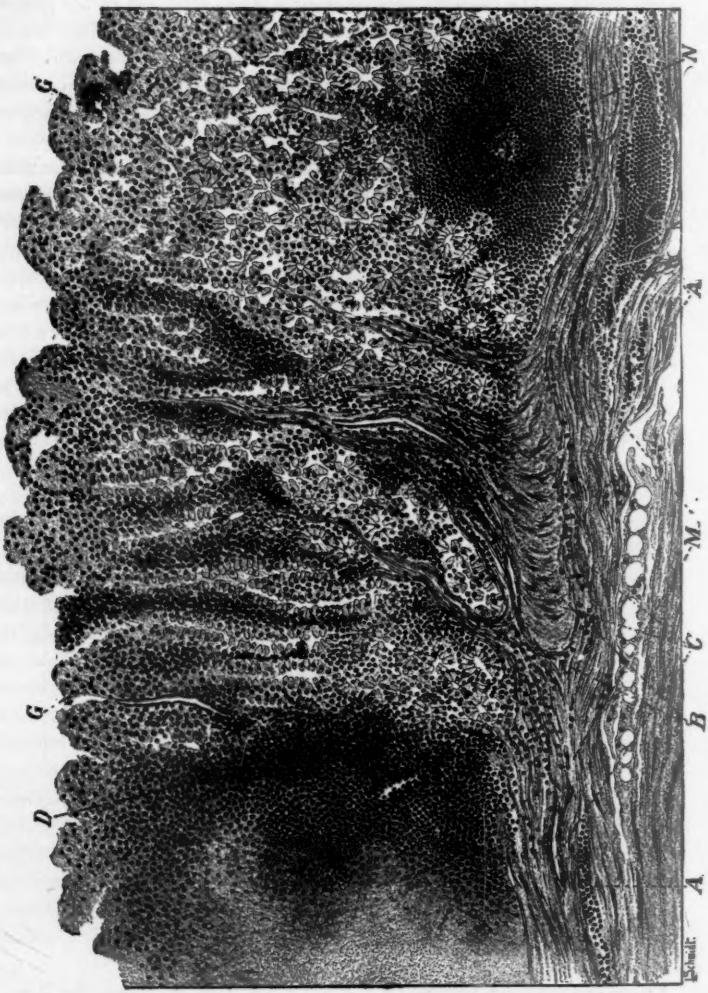


FIG. 1.

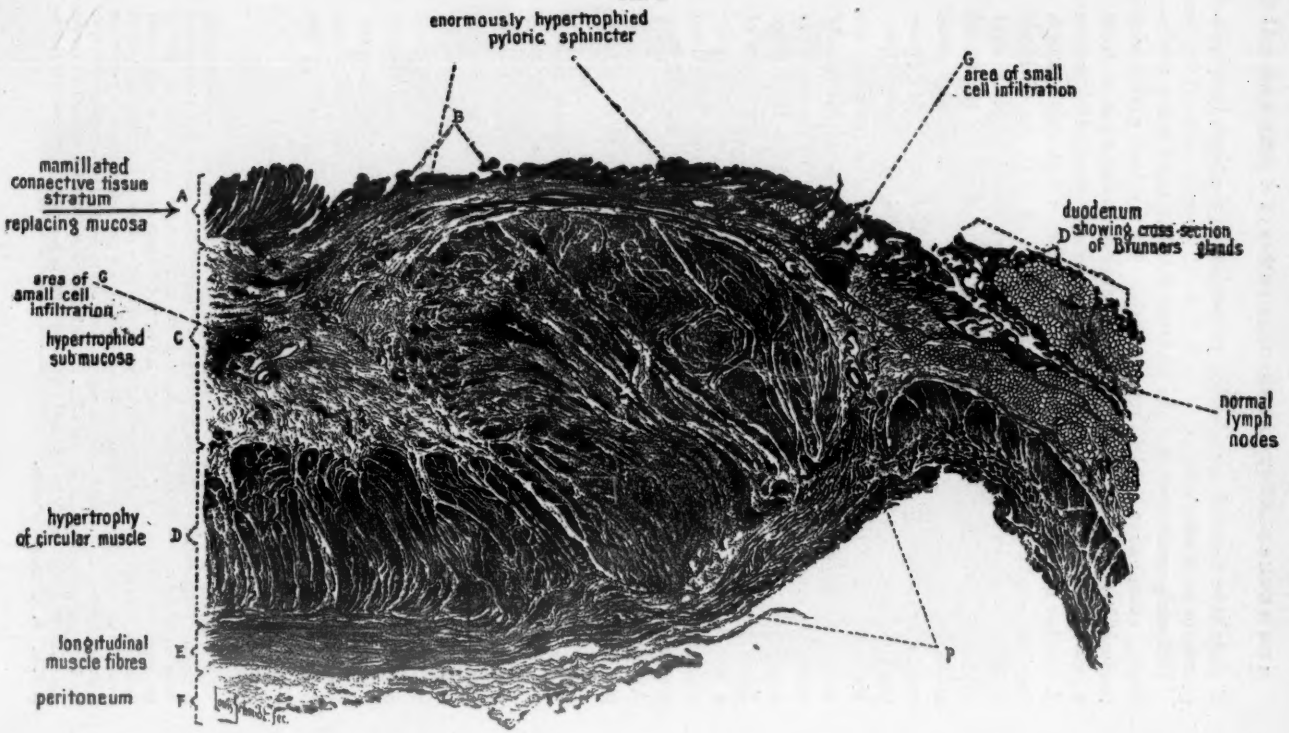
if they have assumed considerable proportions in the liver, lymphatic glands, or at other places.

Difficulties may arise for the macroscopic, but not for the microscopic, examination, which reveals either the typical granulation tissue, if it is in a fresh condition, or if retrogression has set in it shows a caseous and fatty detritus surrounded by strongly marked cicatrices. A number of German pathologists have described a syphilitic sarcoma (sarcoma syphiliticum, see Hansemann, loc. cit., s. 194), the diagnosis of which is more difficult, since the clinical development is very similar to that of a malignant tumor. Beginning at one place and afterward appearing at others, recurring after extirpation, its frequently great resistance to syphilitic remedies—all these puzzling factors should cause the histologist to approach these tumors with a critical conservatism. Macroscopically the extensive fat metamorphosis and the caseation are very noticeable. And even if all the tumors are not in this state of regressive metamorphosis, there are nevertheless some which are thus constituted, and which finally break up entirely into a fatty pulp. Those places which still remain usually show the perfect structures of a soft large-cell sarcoma, in which the very abundant, finely reticular tissue strikes the eye, and in which the larger cells are embedded somewhat loosely, so that they can be brushed out to a certain extent. At the same time single tumors can usually be found which do not possess this great cellular development, and which appear as typical gummas. From this it follows that, after some practice, the case as a whole will be recognized as syphilitic, but that single extirpated tumors often do not permit a diagnosis which consequently reaches only a certain degree of probability. Naturally only the large round-cell sarcomas can give rise to error, not the spindle-cell sarcomas, the melanosarcomas, and the sarcomas with a specific parenchyma, as the osteosarcomas, chondrosarcomas, gliosarcomas, and those with giant cells. It is stated by some authors[1] that cancer-like formations may also arise from syphilis, or that there are actual syphilitic cancers. Hansemann denies this altogether, and asserts that these carcinoma-like developments could only be cases of atypical proliferations of the

[1] Hansemann. Loc. cit.

FIG. 1.—Syphilitic neoplasm of the stomach (from Hemmeter's *Diseases of the Stomach*, 3d edition). The superficial columnar epithelium and that of the vestibules and upper gland ducts is entirely lost. Enormous small round-cell infiltration has forced asunder the fibres of the muscularis mucosæ at *A*, *A*. At *N* and *D* the round cells have accumulated in large nodes extending from the muscularis mucosæ through the remnants of the glands. The gummatous nodule to the left of the illustration has broken down into a pale homogeneous non-staining granular débris. At *B* large bundles of muscle fibres are forced up into the glandular layer by distorting infiltration and proliferation. Within these bundles are contained numerous small arterioles at *C*. A few remnants of the gland ducts in a state of necrobiosis are visible at *G*, *G*. *M*, Muscularis mucosæ.

FIG. 2.


enormously hypertrophied pyloric sphincter
B
G
area of small cell infiltration
mamillated connective tissue stratum
A
replacing mucosa
duodenum
D
showing cross-section of Brunners glands
area of small cell infiltration
G
hypertrophied submucosa
C
normal lymph nodes
hypertrophy of circular muscle
D
longitudinal muscle fibres
E
peritoneum
F
P

epithelia in syphilitic ulcers or tumors. The relatively frequent occurrence of real cancers in individuals who are or were syphilitic, he assumes to be only a chance coincidence.

It is evident from the above (Fig. 2) that the exclusion of infectious tumors is not always an easy matter, but let us assume that these have been excluded, and we find that we are dealing with a real tumor, the most important question arises for the examiner, "Is the tumor malignant or not?"

The most reliable criterion of malignancy is the metastases. We must, above all, hold to that idea of malignancy which is given by the occurrence of metastases, and bear in mind that the metastases may commence before separate knots have been formed, distinct from the primary tumor, but according to Hansemann, we may speak of metastases even when single, viable cell groups have separated from the primary proliferation and lie embedded in the deeper tissue, that is, when the tumor has become *heteroplastic*, in the sense of Virchow. (Heteroplasia, from ἕτερος, other, and πλασις, shaping, meaning the presence of tissue or cells that may or may not be normal in a part where they do not normally belong.) But even this is not necessary for the diagnosis of gastric malignant neoplasms. On the contrary, it is sufficient if it is proved that the proliferation has broken beyond the boundaries of the starting tissue (gland cells in adenocarcinoma) downward. To be sure, it is hard to determine in many cases where the boundaries of many organs are to be looked for, since these boundaries may be displaced with the increase of the proliferation. A sarcoma of the tonsils and of the lymphatic glands has gone beyond the boundaries of the organ when it grows into the muscular tissue; a sarcoma of the bone-marrow has done the same when it has pierced through the bone. But a wart on the skin grows not only upward and outward, but also downward; it displaces the lower boundaries of the epidermis, and yet it is not a cancer. We should rather keep here to the criterion of the disintegration of the neighboring parts. If the epithelia possess a membrana propria, this disappears. The lymph spaces approach the tumor cells in an entirely open manner, and it can be seen that these heteroplastic cells are pressed into the lymph spaces. This crowding of heteroplastic cells into the lymph spaces and the ensuing new formation of connective tissue and vessels give rise to the alveolar structure—beneath the original surface. This heterotopia has already been emphasized by Virchow,[1] and is considered the most deciding

[1] Virchow's Archiv, Bd. iii.

FIG. 2.—Hypertrophic stenosis of the pylorus from chronic syphilitic gastritis. Section through hyperplastic pylorus and comparatively normal duodenum.

factor in the diagnosis. While I agree with Ribbert and Hansemann in assigning considerable importance to metastasis as a criterion of malignancy, I do not wish to uphold this phenomenon as an invariable test, for even benign tumors, myomas, and chondromas have been known to form metastases.[1]

The most experienced teachers of pathological histology advise examination of the tissue first with low powers of the microscope. In examining sections of neoplasms of the gastro-intestinal tract, the *muscularis mucosæ* is, as a rule, my landmark. Any proliferation of epithelial cells around a gastric ulcer, for instance, which has extended downward along the peptic ducts and broken through the "muscularis mucosæ" I am in the habit of considering malignant. At the same time I admit that gastric adenocarcinoma may develop from cells normally located, and that dislodgement of cells from their normal site is not necessary for the origination of such a cancer, though it may be the rule.

Hansemann asserts that by a study of the pathological reversion in neoplastic cells, which he has termed "anaplasia," it is possible to recognize malignancy in the absence of heterotopia. In the following I shall try to represent the characteristics which he conceives are essential to make up anaplastic cells. He has undoubtedly merited the gratitude of pathological histologists for having first called attention to the "undifferentiation," though it is occasionally not observable in the original or mother tumor, and in my experience, as a rule, becomes evident, if at all, in the metastases. It is well to bear in mind that during anaplasia no new kind of cells are developed which might be considered peculiar to themselves, but that it is essentially a return or reversion to a stage of earlier development, from which Ribbert believes[2] that they develop still further in the direction of embryological growth. Pathological reversion to an embryological state and anaplasia have the same significance for proliferation of tumor cells. Concerning Hansemann's statement that malignant tumors of the stomach have occurred which caused death and no heterotopia was recognizable microscopically, I must say that in a very large experience with gastric malignant neoplasms no such case has come to my observation. All gastric malignant tumors examined at operation or autopsy by myself showed heterotopia.

The most difficult histological diagnosis of stomach tumors has, in my experience, been encountered in deciding between chronic hyperplastic gastritis and scirrhus. On two occasions I was unable to make this differentiation from pieces excised from gastric neoplasms during operation. In one case operated by Dr. L. McLane Tiffany the gastric

[1] Lubarsch. Zur Lehre v. I. Geschwülsten, p. 263. [2] Pathologie, s. 613.

neoplasm was not larger than the size of a half-dollar, had thickened the entire pyloric ring, and no metastases were visible as far as the abdominal incision permitted me to see. I favored the view that it was a hyperplastic gastritis. Yet at the autopsy it proved to be a scirrhus, for the fibrous, dense stroma contained a few narrow rows of cancer cells (cancer bodies) running parallel with the dense connective tissue. There was also a metastasis in the liver, larger and softer than the original tumor, and five small nodules in the omentum.

The proliferation of the great majority of cancers in the digestive tract starts from the surface, cylindrical, or columnar epithelium, or from the gland cells. Hansemann asserts that carcinomata of the stomach occur, which cause death without having ever penetrated downward. This author also refers to the extensive carcinomatous destruction at the vaginal portion of the cervix, in which all the cancer cones are still connected with one another, and with the lower surface.[1] Twice I observed the same condition in the intestines, others report this state in the peptic ulcers and in cancroids of the œsophagus. It represents a growing down into the deeper layers while maintaining a real continuity with the surface, not only a microscopic continuity. These are real cancers without heterotopia of the cells. If a diagnosis is to be made in such a case we cannot start from the coarse, structural image, but we must view the morphological change in the cells themselves, if such change is at all discoverable. At this stage of affairs Hansemann insists that we must investigate the anaplastic changes in cells if we want to attain a diagnosis at all in such cases.

To recognize the condition of anaplasia in tumor cells is not as easy a matter as Hansemann would persuade us to believe. It was emphasized before, in this connection, that only the positive result is of any importance for the diagnosis, that the absence of certain characteristic appearances does not necessarily exclude cancer. To recognize anaplasia of cells we must bring in array all possible factors which may influence the process. It is especially necessary for this that the histogenesis of the tumor be determined or that this be reasoned out from its form and location. It is also necessary to know accurately from experience all the morphological, physiological, and especially the formative properties of the parent cells. Then the form of the tumor cells should be especially compared with those of the parent cells. Then we should try to form a conception of the life-history of the tumor cells, how they appear in their youthful condition, how they develop, if and how they finally die. This should also all be compared with the fate of the parent cell. Then we should try to

[1] Cullen. Carcinoma of the Uterus.

find out as much as possible about the functions of the cells, from the histological picture—whether the secretion is a normal one, an increased, weakened, or changed one. Furthermore, if they form an intercellular substance, and if this corresponds to the normal one of the parent tissue, that is, fibrous, osseous, or cartilaginous, lumpy or otherwise.

Finally, we come to the nucleus—segmentation figures—the karyokinesis or mitosis. As to the pathological forms more directly, I may repeat that the simple occurrence of hyperchromatic, abortive, or other pathological mitoses is of no importance at all for the diagnosis. If, however, such pathological mitoses are abundant and considerably heaped together, we can assume with tolerable certainty that a malignant tumor exists. This is not a physiological necessity, but a matter of experience, that a larger collection of pathological mitoses (nucleus-segmentation figures) occurs only with malignant tumors. It would, therefore, be theoretically possible that this should occur also in cases of benign tumors, but Hansemann claims to have never observed it. The presence of atypic mitoses in benign tumors is reported by Lubarsch (loc. cit.), and undeniably does occur, but it is the exception, not the rule. In all benign tumors, however, the pathological mitoses are few and isolated. Of course, there are malignant tumors in which very few pathological mitoses are found. The number of the mitoses is of no importance whatsoever for the kind of the tumor, and it would be entirely erroneous to desire to conclude from a collection of mitoses at all that the proliferation is malignant. In reality it denotes a rapid increase in the cells, and is everywhere found where tissue grows rapidly or is rapidly degenerated. A small number of mitoses are also sometimes seen in malignant tumors if a momentary stoppage of the growth has taken place.

The most important diagnostic factor of the mitoses has always been the variation from the normal forms of the mother-tissue. If such differences are found, it is a sure sign of anaplasia. To avoid every misunderstanding, however, Hansemann repeats that it is not the question of one figure or of another, but that a change must have taken place in the physiological mitoses throughout if one wants to decide upon an anaplasia. Only the positive result is of value, the negative has no value whatsoever.

All observations of mitoses have reliable value only when made on faultless material. In specimens obtained by washing the stomach, post-mortem changes frequently make this part of the examination impossible, since the nuclear forms change very easily, and also because their number decreases. But by my method of gastric curettage with a stomach-tube (see Hemmeter, *Diseases of the Stomach*, third edition) specimens may be obtained in a fresh condition, showing in some cases

of gastric carcinoma an abundance of abnormal mitoses. At times, however, and more frequently than generally supposed, pieces showing the actual structure of carcinoma are thus gained.

In many cases the determination of the regressive metamorphoses in the parenchyma may be of importance. All malignant tumors show an especial tendency toward disintegration, and especially cancers, which are situated on the surface, almost always form ulcers. But it must be remembered that such a metamorphosis also occurs in benign tumors, for instance, in myomas, and that the syphilitic tumors undergo an excessive fat metamorphosis accompanied by caseation. This property of malignant tumors is only to be utilized for the diagnosis in connection with the entire structure, and in itself is not conclusive. Only in those cases in which it presents a form which is not peculiar to the parent cells under other conditions does it point to the anaplastic changes of the cells.

Accordingly, Hansemann emphasizes that it is possible to diagnose malignancy from the parenchyma of tumors alone. This is not true for all cases, but for many. Practically there is little opportunity or necessity to make use of studies of anaplasia for the diagnosis of neoplasms of the digestive tract. Moreover, the importance of these changes as diagnostic and pathogenetic factors has been lessened by the critical logic of Lubarsch (loc. cit.), Ribbert (loc. cit.), and others. Specimens gained at operation or autopsy, as a rule, permit of a diagnosis, from the presence of heterotopia and metastases, in my experience. The stroma in itself, however, furnishes no argument for the diagnosis of malignancy, and only when it accompanies the parenchyma does it complete the histological picture from which was derived the definition of cancers and sarcomas. It must be repeated here again specially that the infiltration of the stroma does not form a basis for the diagnosis, since there are both cancers and benign neoplasms without such infiltration, and benign inflammatory proliferation with it. A number of times I have observed that in portions of adenocarcinoma of the stomach that were derived from the glandular layer the stroma was made up of connective tissue, but in pieces from the muscularis the stroma was composed entirely of muscle fibres. This shows that the stroma may vary, according to the heterotopia.

Cancers of the mouth, œsophagus, stomach, intestines, and rectum, therefore, are diagnosed: 1. According to their structure. 2. Their topographical conduct toward neighboring tissues. 3. According to the properties of the parenchyma in comparison with the parent tissue.

On the other hand, the concept of sarcoma is somewhat more arbitrary. There are some, to be sure, which we recognize in the same way as cancers are recognized. These are the neoplasms presenting a specificity in the structure of their parenchyma—the chondrosarcomas,

osteosarcomas, myogliosarcomas, and lymphsarcomas. The others, however, are characterized by their structure alone, by the development of their cells and the formation of an intercellular substance. In four cases I encountered sarcomata of the intestines with indifferent spindle or round cells, the comparison of which with the parent cells was impossible; malignancy had to be determined by the topography of the same, that is, from the heterotopic development of the tumor. The advance of the tumor into neighboring tissues is what gives the decision alone, for the diagnosis of malignancy. The more the tumor is developed, the easier the diagnosis. But just at the first stages, when a decision would be most valuable, the characteristic criteria very often are lacking, and we must content ourselves with a probable diagnosis. M. Borst suggests that it will always be better in such cases to make the prognosis worse rather than better. This statement cannot well be harmonized with that of Virchow, that there are neoplasms of such perplexing structures that they may be designated as sarcomas or something else, just as the observer pleases.

While we can yet form diagnoses, with tolerable certainty, of the two forms of tumors, cancers, and sarcomas, the malignancy of complex tumors is exceedingly uncertain, and only to be diagnosed from the anatomical picture when its malignancy has already become manifest, that is, when real metastases have been developed. It is well known that all these tumors have occasionally become malignant. Why, however, they should at one time remain confined to their primary position, at another go beyond it and form metastases, is entirely beyond our discernment. No such judgment is possible from the histological structure. However, mixed tumors of the kidneys and uterus are always malignant, and such tumors in some other organs always become malignant if they exist for some time. To this class belong the papillary cysts of the kidneys and ovaries, besides the tumors growing from parts in the neighborhood of the kidneys to the kidneys themselves, which finally spread to the vena cava and form metastases throughout the body.

One of the most striking phenomena of all malignant tumors is the very widely differing degree of their malignancy, which fact has been mentioned several times. There are even cancers which exist ten years or more without revealing their malignancy in the shape of metastases. The question may be asked, "Can any deductions be made from the histological structure concerning the degree of malignancy?" The study of anaplasia might furnish a starting point for this, for from it we see without a doubt that those cells which have most increased in the power of independent existence must also most easily give rise to the formation of metastases. Since this is expressed by the degree of variation from the parent tissue a certain parallelism

ought to exist between the degree of anaplasia and the degree of malignancy. In order to control this question, Hansemann made the following two series of observations. At one time the metastases and recurrences were compared with the primary tumors, and it was seen that they showed the same or a greater degree of anaplasia than the primary tumors, never a less degree. Furthermore, tumors with very extended dissemination through the entire body show strong anaplasia; such with uncommon local extension, without dissemination, show slight anaplasia. There are few exceptions to this law. For theoretic consideration Hansemann considered this law proved in general; the greater the anaplasia the greater the tendency to form metastases. The second series of observations was concerned with carrying this theory over into practice. For this purpose he made notes as to the degree of anaplasia and the consequently expected tendency to recurrences of metastases. These prognosticated data were, as far as possible, controlled and compared with the later real course of events. The result was as follows: If the tumor was found to be very anaplastic, recurrences and metastases were hardly ever lacking. If, however, the tumor was but slightly anaplastic, either no recurrence or metastases occurred, or they developed with greater or great anaplasia. It has not yet been discovered on what fact it depends that a tumor becomes more anaplastic, and, therefore, the prognosis in all such cases is doubtful. Here, also, strong anaplasia gives more information than anaplasia which is slight or not discoverable at all.

My personal experience, as far as the practical application of this theory to the relative frequency of recurrences or metastases in cases of gastro-intestinal neoplasms is concerned, is that in all of my cases of this sort recurrences were recorded in from one to three years after the operation. I mean in all cases, of course, the future history of which could be traced up; that is, in forty-eight out of sixty operated cases.

I included in this list only such cases at which no metastases were visible at the operation.

The factors necessary to constitute anaplasia are not readily comprehended or recognized. 1. The establishment of the histogenesis of the tumor from its form and localization. 2. An accurate knowledge of the morphological, physiological, and formative peculiarities of the mother cells. 3. Comparison of the forms of tumor cells with those of the mother cells. 4. The biology—*i. e.*, life history of the tumor cells—their youthful appearance, their growth, their decline and death. 5. Determination of the function of the tumor cells, whether they secrete or not. 6. Character of the intercellular substance and its comparison with that of the mother tissue. All of these factors demand much critical discernment, large casuistic experience,

and patient judgment, so much so that the study of anaplasia is practically impossible outside of pathological laboratories.

Before concluding I desire to call attention to the gastric adenoma originating on the basis of a gastric ulcer. It is a well-known fact of pathology that the human gastric or peptic ulcer may become transformed into an adenocarcinoma apparently spontaneously. This process has been described in a classical investigation by Hauser.[1]

In 1900 I paid a visit to Prof. Hauser's laboratory at the University of Erlangen, and was fortunate enough to see some of his original preparations and sections. I have also had a rather exceptional clinical experience with cases of gastric ulcer in the human being, which I had studied in one case for over two years, during which it presented the typical clinical history of gastric ulcer. Then followed the clinical history of gastric carcinoma and death.[2] Naturally the question arose in my mind: "If gastric ulcers could be produced experimentally in animals, their transmission into possible adenocarcinoma might be studied at will, or the transformation into adenocarcinoma might be attempted by the transplantation of malignant neoplasms derived from other animals of the same species, or by the injection of sterilized filtrate of ground-up adenocarcinoma of other animals of the same species into the tissues surrounding the edge of the ulcer."

Among the causes that are definitely ascertained as contributing to the bringing about of gastric ulcer are especially three:[3] 1. Impaired vitality or injury to the mucosa of the stomach. 2. Hyperacidity or super-secretion of gastric juice containing an excess of HCl and proteolytic ferments. 3. An altered state of the blood.

All three of these factors may be produced artificially, and when we do produce them in the laboratory on animals, peptic ulcers arise in such a large proportion of the animals thus experimented upon that we have a right to conclude that they are directly attributable to the method pursued. When I speak of peptic gastric ulcer I mean the typical "Chronisches magengeschwür" as described by Hauser.[4] For defects can be produced in the dog's stomach, for instance (and the literature on the experimental production of gastric ulcer proves this conclusively), which are not true gastric ulcers, but simply ulcerating lesions which heal rapidly. Gustav Fütterer[5] has produced such lesions by applying to the stomach caustics, ligating the gastric arteries, cutting off the supply of nutrition by stitching large portions of the mucosa

[1] Das chronische Magengeschwür, sein Vernarbungs-Process und die Beziehung zur Entwicklung des Magencarcinom, Leipzig, 1883; and Das Cylinder-epithelcarcinom des Magens und des Dickdarms, Jena, 1890. See also Hemmeter, New York Medical Record, 1897, vol. lii., p. 365. Also Hemmeter, Diseases of the Stomach, 3d ed., p. 560.

[2] Hemmeter. New York Medical Record, 1897, loc. cit.

[3] Hemmeter. Diseases of the Stomach, 3d ed., p. 491.

[4] Loc. cit.

[5] Ueber die Aetiologie des Carcinoms, p. 115.

with catgut sutures. Defects resulted, but they all healed in a short time. Even when one-third of the quantity of the animal's blood was withdrawn, such defects had healed in two weeks. In 1896 Silbermann produced gastric ulcers in dogs by either tying the gastric arteries or causing emboli in them by injecting suspensions of lead chromate. Thereafter he injected hæmoglobin and pyrogallic acid (acid pyrogallic 0.1 to 0.14 per cent.). When Fütterer resected the gastric mucosa, as stated before, and made injections of pyrogallic acid, he could confirm Silbermann's results and produced ulcers of the stomach which in every histological detail corresponded to the chronic gastric ulcer of the human being. By this method, employed by Fütterer and Silbermann, I was successful in producing experimentally gastric ulcers. In one series of experiments eleven dogs out of thirty operated upon developed typical gastric ulcers.[1]

Now I had a method by which this characteristic lesion could be produced experimentally. The next question was: "Could these lesions be in any way experimentally transformed into adenocarcinoma of the stomach, or would they become transformed into adenocarcinoma spontaneously, as has been definitely known to occur in the human being, and as Fütterer has observed, to occur in a rabbit in which he had artificially produced a gastric ulcer by the method described?"[1]

Hauser has described a structural characteristic of the adenocarcinoma of the stomach, which has developed on the basis of a gastric ulcer, and which is not observed in ulcerating carcinomas not developed on this basis. This characteristic, by which the so-called "ulcus-carcinomatosum" can be recognized, consists in a very peculiar behavior in the fibres of the chief muscular layer of the stomach, and also of the fibres of the muscularis mucosæ. This peculiar behavior of the muscularis consists in an oblique ascension of the fibres of the true muscular layer, and a descension of the fibres of the muscularis mucosa, the fibres of both muscular layers converging toward and fusing into each other in front of the edge of the ulcer, which is here composed mainly of connective tissue. The true muscularis bends upward in continuity, and the border of the ulcer, which is composed of very dense connective tissue, is limited by the lower surface of the turned-up true muscularis. Another feature of the ulcus carcinomatosum is that a section made perpendicular to the surface of the stomach, and through the entire bed of the ulcer, almost invariably exhibits the general outline of a fish-hook.[2]

For the causes which bring about this fish-hook formation (see Fütterer, loc. cit., p. 112), the main reason being that the lower edges of

[1] Hemmeter. Intracellular Catalytic Processes in Pathogenesis of Malignant Neoplasms. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, April, 1903.

[2] Loc. cit., p. 152. [3] Hemmeter's Diseases of the Stomach, 3d ed., Plate IX.

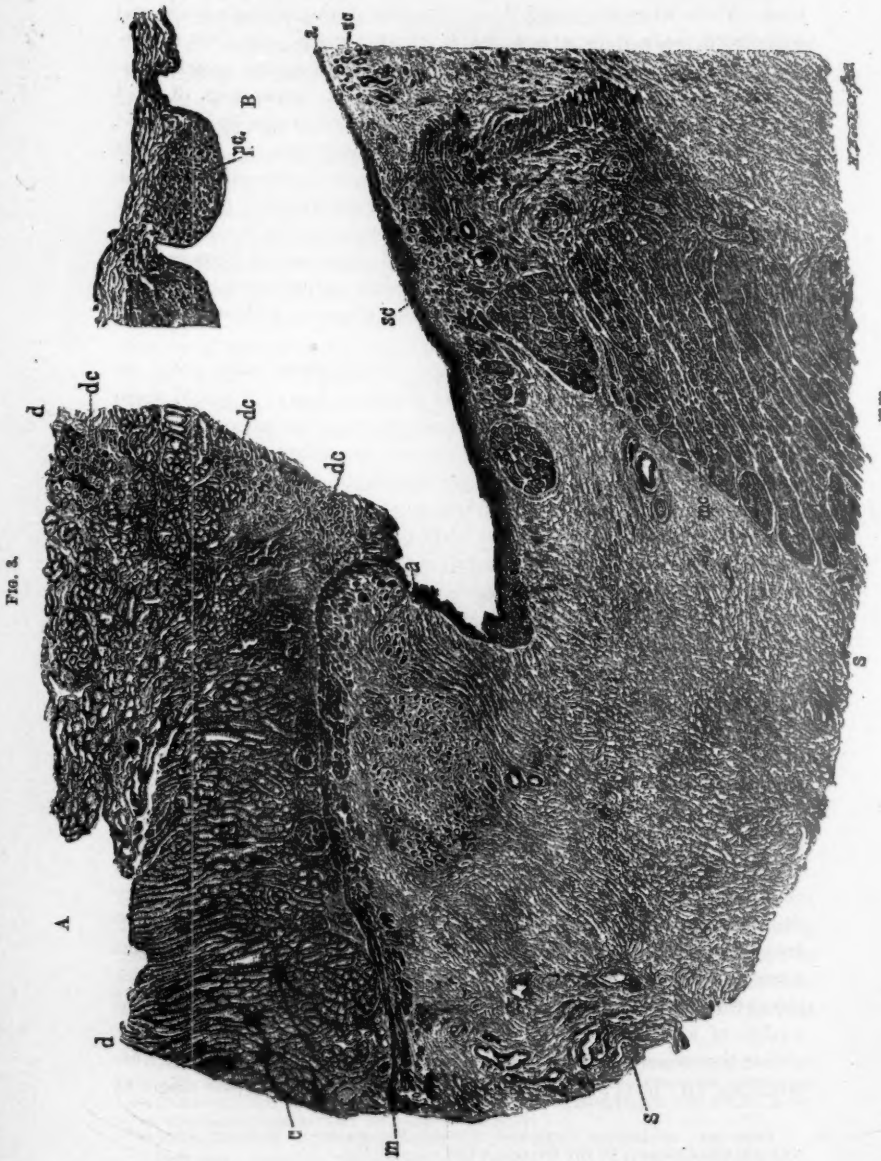


FIG. 2.

the ulcer, near the pylorus, are, during the efforts of the gastric peristalsis to evacuate the gastric chyme into the duodenum, exposed to the most mechanical irritation, and accordingly Fütterer has shown that adenocarcinoma, if it develops from an ulcer, always develops from the lower edge. This location for the development of gastric ulcer has already been emphasized in a publication by Dr. Delano Ames and myself,[1] but it is the desert of Fütterer to have emphasized this point as an etiological factor in the causation of ulcus carcinomatosum.

I should add that in repeating the experimental production of gastric ulcer according to the methods of Silbermann[2] and Fütterer,[3] I not only produced mechanical defects and injected pyrogallic acid in the method described, but I maintained a very high acidity for free HCl in the gastric chyme of the dog by supplying this acid in his food, and also pouring it into his stomach through a soft rubber tube. In February, 1900, I came into the possession of a mongrel fox-terrier that persistently vomited his food. The vomit contained no free HCl nor ferments, and at the autopsy a cancer of the stomach was found near the pylorus. Transplantations with this material were made into the stomach of other fox-terriers, but I was not successful in producing a gastric carcinoma in any of these other animals inoculated.[4] Dr. Leo Loeb has undoubtedly succeeded in transplanting a sarcoma from one rat thus affected to a large number of other rats of different age and sex.[5] In my previous publication[6] I stated that, as far as I knew, Loeb's work had not been confirmed. In a private communication Dr. Loeb informs me that Ehrlich and others confirmed his results. A sterile extract of part of this canine carcinoma was also made and preserved for inoculation and injections into the stomachs of dogs in which I

[1] New York Medical Record, September 11, 1897.

[2] Loc. cit. [3] Loc. cit.

[4] Sepsis followed a large proportion of the operations, and I could not secure the aid of a competent surgeon who would do these operations aseptically for me.

[5] Journal of Medical Research, vol. vi. No. 1.

[6] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, April, 1903.

FIG. 3.—Ulcus carcinomatosum of the pylorus. *A.* A section through the wall of the stomach, showing the edge and a portion of the base of the ulcer. Objective, two-thirds; eye-piece, two inches. Stained with hematoxylin and eosin. The drawing is built up from a series of microscopic fields. × about 15 diameters. *d.* Mucous membrane. *m.* Muscularis mucosæ. *s.* Submucosa. *a.* Base of the ulcer. *mm.* Muscle coat of the stomach. *mc.* Groups of cancer cells between the bundles of muscle fibres. *dc.* Groups of cancer cells in the edge of the ulcer in the mucous membrane. *sc.* Groups of cancer cells in the submucosæ. *a.* Necrotic membrane lining the base of the ulcer.

B. A small nodule from the serous coat of the stomach over the base of the ulcer. Objective, two-thirds; eye-piece, two inches. Stained with hematoxylin and eosin. × about 15 diameters. This figure gives a good idea of one of the nodules in the serosa. It is composed entirely of a collection of groups and masses of cancer cells so closely packed that the outlines of the individual cells cannot be made out. Except for these nodular thickenings, the serosa was not altered. *pc.* Cancer masses in peritoneal coat.

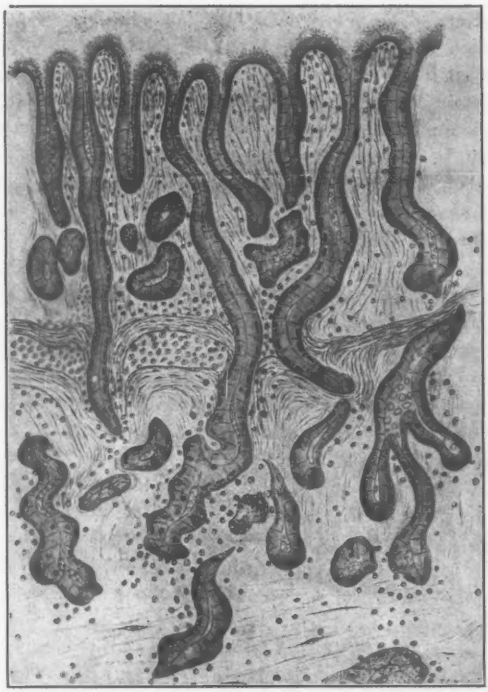
expected to cause gastric ulcers. A very small quantity of filtrate (148 c.c.) was thus saved, as the largest part of the tumor had been used for transplantations.

FIG. 4.

Normal gastric mucosa of dog. *A.* Glandular layer. *B.* A homogeneous layer characteristic of canine and feline stomachs. *C.* Muscularis mucosæ. *D.* Submucosa with few muscle bundles.

In the meanwhile the publications of Fütterer[1] appeared, and it occurred to me that possibly my failure to successfully transplant canine carcinoma was due to the fact that the stomach of the animal into which the inoculation was made was not in a susceptible condition, and that it must be transformed into such a condition by a

FIG. 5.



Adenomatous proliferation at edges of experimental gastric ulcer caused by injection of sterile and cell-free extract of gastric adenocarcinoma of a dog. *A.* Surface of gastric mucosa. *B.* Peptic gland ducts. *C.* Muscularis mucosæ. *D.* Proliferating gland ducts that have broken through the muscularis mucosæ. *E.* Gland cells in submucosa.

previous injury. This previous detriment to the tissue, in order to secure successful transplantation of the tumor, in the case of rats, has not been found necessary by Leo Loeb[2] and Herzog.[3] I did not succeed in securing another dog affected with carcinoma of the stomach

[1] Loc. cit.
[2] Journal of Medical Research, vol. viii. p. 44.
[3] Ibid., p. 74.

until after I had read Fütterer's work in 1901, and this second dog presented an adenocarcinoma which developed spontaneously at the edges of an experimental gastric ulcer. It presented the behavior of the two layers of the gastric muscularis as first described by Hauser, had a distinct fish-hook form, and exhibited heterotopia of gastric glands as first described by Virchow[1] and Hansemann.[2] This gastric cancer reached the size of a walnut, and two metastases were found in the peritoneum.

The practical deduction for the histological diagnosis for malignant tumors which can be made from the studies of Hauser, Fütterer and myself, is that it is possible to diagnose a carcinomatous ulcer of the stomach histologically by the characteristic behavior of the true muscularis and muscularis mucosæ, and by the form of a fish-hook presented in outline in a section through the edge of the ulcer nearest the pylorus. By these striking features I believe we are justified in concluding that any gastric adenocarcinoma presenting them has been developed on the basis of a pre-existing gastric ulcer.

In the April number of this JOURNAL I have reported the results of my experiment attempting the transformation of experimental gastric ulcer into adenocarcinoma by injecting the extract of a canine gastric carcinoma into the edges of the ulcer. Very striking adenomatous proliferations can be produced in this way, but they cannot be called carcinomas. I include here the photographs of some of the sections, showing these experimental adenomas.

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[1] His' Archiv, Bd. iii.

[2] Loc. cit., p. 195.

SYPHILIS OF THE TRACHEA AND BRONCHI.

AN ANALYSIS OF 128 RECORDED CASES AND REPORT OF A CASE OF SYPHILITIC STENOSIS OF THE BRONCHI.

By Lewis A. Conner, M.D.,
PROFESSOR OF CLINICAL MEDICINE IN THE CORNELL UNIVERSITY MEDICAL COLLEGE; ATTENDING PHYSICIAN TO THE HUDSON STREET HOSPITAL, NEW YORK.

The observation of an obscure case of pulmonary disease which the autopsy showed to be one, primarily, of syphilitic stenosis of both bronchi, was the occasion of an investigation into the literature of the subject of syphilitic manifestations in the bronchi.

It was soon found, however, that a separation of the cases of syphilis of the bronchi from those of syphilis of the trachea could not with propriety be made, since both in their clinical and in their pathological aspects the conditions seemed identical, and since both were frequently found associated in the same subject. Accordingly, an attempt has been made to collect and analyze as many as possible of the recorded cases of syphilis of the trachea and bronchi. A few cases have been omitted in which there seemed reasonable doubt as to the accuracy of the diagnosis or in which the records were too incomplete to be of value.

No instance of the occurrence in the trachea or bronchi of the initial lesion of syphilis has, so far as I can discover, ever been recorded. Observations, also, as to the appearance in the trachea of the *secondary* manifestations are confined to a very few cases. Schroetter[1] asserts that he has never seen condylomata, macules, or papules in the trachea. Lancereaux[2] states that secondary lesions are very rare, and are seen as disseminated areas of hyperæmia of the mucous membrane in the form of violet-red spots which disappear rapidly. Morell Mackenzie[3] has seen mucous patches in the trachea only five times.

Not only are these tracheal manifestations of secondary syphilis extremely uncommon, but they are also fleeting in character and have apparently little or no clinical significance. For all practical purposes, then, the lesions of syphilis in the trachea and bronchi may be said to be confined to those late and severe manifestations known as tertiary.

These tertiary syphilitic conditions of the trachea and bronchi, however, have, it seems to me, an importance quite out of relation to their great rarity for the reasons, first, that in almost every recorded case they have gravely threatened life, and in most cases have actually caused death; and, second, that from a study of the cases it seems quite evident that a very large proportion of them are curable if they can but be recognized and treated early.

Historical. Although the occurrence of ulceration in the trachea, without associated lesions in the lungs or larynx, had been recognized

earlier (Cayol[4]), the first case of disease of the trachea or bronchi ascribed to syphilis seems to have been that of Munk. In the *London Medical Gazette* for 1841, p. 210, he describes the case of a man who had syphilis for eighteen months before death, with various severe manifestations, including ulceration of the soft palate. The necropsy showed many minute ulcers in the mucosa of the larynx, none in the trachea, but many in the bronchi, especially in the smaller ones. In the following year (1842) Worthington[5] reported a carefully observed case of stricture of the trachea which he believed to be syphilitic, and in 1843 O'Ferrall,[6] of Dublin, reported one of extensive lesions of the epiglottis and trachea which were clearly syphilitic, although he does not so state. Both of these latter cases present classical pictures of the disease in both its clinical and its pathological aspects.

In the same year Watson,[7] of New York, recorded a case of syphilitic ulcer of a bronchus, with sudden, fatal hemorrhage.

Within the next few years several cases were described in Germany. In 1848 Waller[8] reported four cases. A decade later three cases were observed in Paris (Moissenet,[9] Vigla,[10] and Charnal[11]), and from that time on cases multiplied. Wilks[12] (1863) recorded four instances, and Türck[13] five more. Gerhardt,[14] in 1867, reported five cases and gave a summary of the clinical features which, even at the present time, can hardly be added to or improved upon.

In 1878 Vierling[15] was able to collect and tabulate 46 cases. I have now endeavored to bring together all such of the earlier cases as are not included in Vierling's table, and as many as possible of the cases reported since that time. These 82 cases, including my own, have been tabulated and analyzed, and the results are here recorded, together with a list, in chronological order, of the cases and a very brief statement of the salient features of each. These cases will be referred to by their Roman numerals. Reference to Vierling's table will be indicated by the letter V. before the number of the case.

Morbid Anatomy. In 117 of the 128 cases of both series, the character and seat of the lesion could be determined; in 97 by autopsy, in the remaining 20 by laryngoscopical examination. The lesions found, while varying exceedingly in character and extent, may be conveniently grouped into four classes:

1. Gummatous swellings—circumscribed or diffuse.
2. Ulcers—single or multiple.
3. Endotracheal connective tissue new-growth—(*a*) distinct scars; (*b*) diffuse thickening.
4. Fibrous peritracheitis.

1. Of the first class (*gummata*) there were in all 20 cases (17 per cent.). Eighteen of these occurred in the present series. Of these 18 cases there were 11 (VIII., XIX., XXX., XLI., XLV., LVI.,

LVIII., LX., LXXIV., LXXVIII., and LXXX.) in which the gummata were circumscribed. Most of these were seen during life by laryngoscopical examination and showed as rounded, red, tumor masses projecting into, and sometimes almost filling, the tracheal lumen.

In 8 cases these tumors were single. One case (LVI.) showed the trachea from the fourth ring downward studded with numbers of irregular nodular swellings, some firm, some soft, some ulcerating. In Case LX. a large gumma was found at autopsy astride the point of bifurcation.

Some of the gummatous swellings (XIV., XXVI., XXXII., XXXVII., LIX., LXX., and LXXII.) instead of being circumscribed extended over considerable areas of the tracheal wall, and sometimes involved the whole circumference. These are described usually as of reddish color and as showing fibrinous or purulent exudate upon the surface. Two of these (XIV. and XXVI.) showed some superficial ulceration.

2. *Ulcers* were found in 27 of the present list of cases, and in 24 of Vierling's (44 per cent. of all cases). In 10 of the 27 cases the ulcers were single. These were, as a rule, large (an inch or more in diameter) and deep. Munk's case (I.) showed many small ulcers in the larynx, *none* in the trachea, but a great many in the large and small bronchi.

The ulcers showed all possible variations in size, contour, depth, position, etc. Usually they were quite deep, penetrating to the submucous layer, and, in many instances, even to the cartilages. In a number of cases these latter have been found necrotic and partly destroyed.

In several cases (*e. g.*, in IV.) fragments of cartilage have been coughed up from time to time.

Perforation of the entire tracheal or bronchial wall occurred in 12 cases and usually with disastrous results. Thus, in 5 of these cases there was fatal hemorrhage from erosion of some large bloodvessel.

In two instances (V. 32 and V. 42) an ulcer in the right bronchus perforated a branch of the right pulmonary artery. Once (II.) there was erosion of a branch of the bronchial artery by an ulcer in the left bronchus. Once (XXV.) the aorta itself was perforated, and once also the superior vena cava (L.). Perforation of the œsophagus occurred in two instances (XXIII. and LVII.). One perforation of the trachea (V. 11) resulted in a fatal mediastinal abscess. In Cases XXXIX. and LII. perforation resulted in peritracheal abscess, which in LII. caused serious tracheal compression below. In Case LXXV. erosion of the tracheal wall resulted in the curious condition known as "tracheocele."

The appearance of many of the ulcers gave evidence of no tendency toward healing. In other cases scarring was in process; in still others

(*e. g.*, LVII.) cicatrization was found in the central portions, while at the periphery the ulcerative process was active. The edges of the ulcers in some cases were thickened and everted; in others undermined and overhanging. In general the ulcers showed a tendency toward circular outlines.

3. *Endotracheal Fibrous Tissue.* (*a*) Distinct scars were found in 25 of the present series of cases, and in 22 of Vierling's—in all 40 per cent. These showed the same variations that were found in the ulcers. Most frequently they were radiating or star-shaped. Often they formed prominent bands of dense tissue projecting into the lumen and running in various directions — transversely, longitudinally, even spirally. In three cases (XXVII., XXXIII., and XLVI.) they formed distinct membranous bands which acted almost like diaphragms.

In Gleitsmann's case (XXXIII.) laryngoscopical examination revealed two antero-posterior membranous bands opposite the fifth or sixth cartilage, with only a narrow slit between them. The appearance was that of a second glottis. Schroetter (XXVII.) at one time saw a tracheal ulcer with some beginning constriction; two months later the ulcer had healed, but the scar had formed a membranous stricture of extreme grade.

In Case XLVI. Masseï saw in the trachea at the third ring a diaphragm-like membrane, with an oval opening near its centre, admitting only a No. 12 or No. 13 French catheter. This was made of two membranous folds extending from the tracheal walls and uniting everywhere except at the opening.

(*b*) In 13 of the cases (11 per cent.) the lesion found at autopsy was a diffuse connective tissue infiltration of the tracheal or bronchial wall *within* the cartilages—an "obliterative endotracheitis" or bronchitis, with marked diminution of the lumen. This growth involved the whole circumference of the wall and frequently covered several centimetres in length. It is doubtful if many of these thickenings had been preceded by ulceration. The proliferation of connective tissue seems rather to have begun in the submucous coat. In 2 of these cases, at least (XX. and LXXXII.), this fibrous endotracheitis was associated with an extensive peritracheitis as well. As regards the location of these lesions, in 1 case (XVII.) the lower part of the trachea alone was involved, in 4 cases (XXXVIII., XLVII., LIV., and LXII.) both trachea and bronchi were affected, in the remaining cases the lesion was confined to one or both bronchi.

Case LIV., one of inherited syphilis, furnished the most striking example of the condition. The thickening of the wall began abruptly one and a half inches above the bifurcation, with diminution of the lumen to one-third of its normal size by enormous production of fibrous tissue within the cartilages. The epithelial covering was, for the most

part, intact. The same condition was found in both bronchi, the left being almost occluded. Parker, in describing this case, gives to the condition the name "obliterative endotracheitis."

4. Not the least interesting of the lesions of tracheal and bronchial syphilis are the masses of dense fibrous tissue which have occasionally been found to develop *outside* of the cartilaginous rings.

Of this group no examples can be identified among Vierling's cases, but in the present series there were 8 (XX., XXXII., LVI., LXII., LXVI., LXVII., LXXVII., and LXXXII.). Of these all but 1 (LXXVII.) were combined with endotracheal lesions of some sort.

This development of peritracheal fibrous tissue was seen usually about the lower end of the trachea and about the main bronchi. One or all of these tubes would be encased in a dense mass of fibrous tissue which frequently involved other adjacent structures and in particular the recurrent laryngeal nerves.

In 1884 Gougenheim and Leval-Piquechef[16] called attention to the chain of small lymph nodes lying deep in the neck between the trachea and the œsophagus, and extending from the larynx to the roots of the lungs, which they found regularly enlarged in tuberculosis. Lécureuil[17] thinks it probable that the peritracheal inflammations of syphilis originate as gummatous processes in these nodes, and that these processes may terminate in dense fibrous tissue entirely surrounding, and often constricting, the trachea and bronchi.

These nodes lie in close relation also to the recurrent nerves which seem so frequently to be involved in the peritracheal inflammation.

Of the 8 cases of peritracheitis here cited, involvement of one or both nerves occurred in 4. In Cases XXXII. and LXII. both nerves were affected; in LVI. the left nerve; in LXVI. the right. In another case (LXIII.), with symptoms of tracheobronchial syphilis, which died without autopsy, the existing paralysis of both posterior cricoarytenoids was attributed to the same cause.

In 3 of the 8 cases the enlarged lymph nodes were recognized as a part of the peritracheal mass. In Case LXVI. a distinct tumor mass 5 x 4 x 2 cm. in size was found partly surrounding the trachea at its upper part, intimately adherent and causing compression and constriction. The mass enclosed the right recurrent nerve, was caseous in places, and had begun to ulcerate into the trachea—the cartilages being bare and eroded. In all the other cases the lesion was much more diffuse and involved the entire circumference of the tube. Two of the observers (LVI. and LXIX.) were struck with the many points of correspondence between these diffuse fibrous strictures of the trachea and the syphilitic strictures of the rectum.

The distribution of the syphilitic lesions may be seen from the following table:

	Cases.	Percentage.
Involvement of upper third of trachea alone	23	19.7
" middle " "	2	1.7
" lower " "	18	15.3
" large area of "	23	19.7
" trachea and bronchi	38	32.6
" both bronchi alone	5	4.2
" right bronchus alone	4	3.4
" left "	4	3.4
Total	117	100

From this table it will be seen that the trachea alone was diseased in 56 per cent. of the cases, the trachea and bronchi together in 33 per cent., and the bronchi alone in 11 per cent. The great rarity of lesions of the middle part of the trachea, as compared with those of either the upper or the lower end, is also made apparent. The table does not, however, bring out clearly the great frequency of lesions at, or just above, the bifurcation.

In almost all the cases in which both trachea and bronchi were affected the tracheal lesions were located chiefly at the lower end—a fact which was noted and emphasized by Gerhardt.[14]

Associated Lesions. *Larynx.* In Vierling's table of cases the larynx is recorded as being diseased in 63 per cent. In the present series it was involved in only 12 per cent. of the cases. This wide difference in the relative frequency of laryngeal involvement in the two series of cases it is difficult to understand. Altogether the larynx was found the seat of syphilitic lesions in 30 per cent. of the cases.

Gerhardt and others have intimated that the association of laryngeal syphilis with that of the trachea was especially common when the tracheal lesions were located at its upper part, but an analysis of the cases hardly supports that view. The frequency was almost as great when the disease was confined to the lower part of the trachea or to the bronchi as when it was at the upper end. Laryngeal involvement was most common, however, among those cases in which the tracheal and bronchial lesions were extensive and severe.

Mouth, Nose, and Throat. Evidence of old or recent syphilis in the palate, nose, or pharynx were recorded in 16 (about 20 per cent.) of the present list of 82 cases. In 6 of the 16 cases (37 per cent.) there were also lesions in the larynx. These figures would seem to suggest that there existed a closer relation between syphilis of the upper air passages and that of the larynx than between disease of the larynx and that of the lower air passages, since in the same series laryngeal involvement was found in but 12 per cent. of the cases of tracheo-bronchial syphilis.

Lungs. Autopsies were made in 56 of the present series of cases and revealed the following pulmonary changes:

In 10 cases (18 per cent.) lesions believed to be characteristic of

syphilis were found (IX., XX., XXVIII., XXXI., XXXII., XXXVIII., LVI., LXVIII., LXX., and LXXIX.). In all of these, except XXXII., there existed interstitial pneumonia of greater or less extent. In 2 cases (XXXII. and LVI.) distinct gummata were found.

Evidences of old tuberculosis were seen in 3 cases (VI., XVII., and XXXI.).

In 19 of the cases pneumonia, more or less extensive, was present.

This in seven instances was described as lobar pneumonia; in the remaining twelve either as lobular or bronchopneumonia. Many of these latter were clearly cases of inhalation pneumonia.

In 3 other cases the lungs showed congestion; in 1 case (XXXIX.) "splenization at both bases;" in 1 case (XXIV.) a gangrenous cavity in the left upper lobe.

Emphysema is mentioned as being present in only 9 of the 56 cases (VI., XII., XVII., XXXIV., XLVII., LI., LV., LXXIX., and LXXXII.). This surprisingly small proportion is probably far from representing the real frequency of emphysema in this condition, as the autopsy reports in many cases were very meagre with respect to the state of the lungs.

Dilatation of Trachea and Bronchi. Among the 97 autopsies of both series dilatation of the trachea was found three times (XVII., XXIV., and V. 15). In each of these instances it occurred *above* the point of stricture.

Dilatation of both bronchi was found in 9 cases (V., VIII., XX., XXII., XXIV., XXXV., XXXVIII., LV., and LXXXII.). One bronchus alone, or its branches, showed dilatation six times (IX., LII., LXX., LXVIII., LXXIX., and V. 12). In other words, there was bronchial dilatation below the lesions in 15 per cent. of the autopsies. The character of the dilatation differed greatly in the different cases. Commonly it was cylindrical and involved not only the main bronchi but their branches as well. In some cases it was described as universal. Less frequently the dilatation was circumscribed and confined to the area immediately below the stricture.

It is not possible here to enter into a detailed discussion of the causation of such bronchiectases. The surprising thing is not that they occasionally occurred, but that they should have been found so infrequently. In all but a few of the cases (perhaps 15 per cent.) obstruction of some sort existed, and yet dilatation was found in only 15 per cent. of the autopsies. That is to say, in not more than 20 per cent. of the cases of permanent stenosis did bronchiectasis occur. It seems evident, therefore, that the tracheal or bronchial stenosis is not the chief determining cause of the bronchiectases found. Much more potent factors, probably, are the changes occurring in the bronchial

wall and in the adjacent lung tissue by which the muscular and elastic tissue of the bronchi, upon which their strength and resilience depend, is replaced by inflammatory tissue.

The autopsy records upon this point are, for the most part, very unsatisfactory, but certain interesting facts are nevertheless developed. Case LXVIII. showed stenosis of the trachea and right main bronchus; there was also interstitial pneumonia of the right lower lobe. Dilatation was confined to those branches of the right bronchus which led to the diseased lower lobe.

Case LXX. Moderate obstruction in trachea; main bronchi not stenosed; interstitial pneumonia in left lung. Dilatation of bronchi running to left lower lobe only.

Case LXXIX. Stricture of left main bronchus; dilatation of bronchus beyond stricture and of branches running to left upper lobe, which is *sclerosed*. Branches to left lower lobe not dilated.

In 1 case (LV.) there was general dilatation of the bronchi, although no stenosis was found and no obstructive symptoms had been present.

INCIDENCE. *Age.* The age in 112 cases is shown by the following table, arranged in decades:

0 to 9 years	4 cases.	40 to 49 years	26 cases.
10 to 19 "	9 "	50 to 59 "	9 "
20 to 29 "	15 "	60 to 69 "	5 "
30 to 39 "	43 "	70 to 79 "	1 "

The youngest case was that of a child, aged fourteen months, reported by Woronichin[18] (V. 45). The age of the oldest case (LXIX.) was seventy-three years.

Sex. Among the 120 cases in which this point could be ascertained there were 68 males and 52 females—57 per cent. and 43 per cent., respectively.

DURATION OF SYPHILITIC INFECTION. The time of the appearance of tracheal symptoms in relation to the duration of the general syphilitic infection varied enormously.

In 31 cases of acquired syphilis in which the point could be determined, the average duration of the syphilis was ten years. The shortest duration was nine months (XI.); the longest, forty-two years (LXVII.). In another case (LI.) the infection had occurred forty years before.

During this intermediate period syphilitic manifestations of some sort are mentioned as having occurred in 20 per cent. of the present series of cases. In over half of these the manifestations were seen in the throat or larynx.

Inherited Syphilis. In 10 cases (VIII., XVIII., XXIX., XXX., LIV., and V. 14, 35, 39, 40, and 45) the lesions were ascribed to inherited syphilis. The average age of these 10 cases was ten

years. The 2 oldest cases were, respectively, twenty and nineteen years (V. 14 and 39).

SYMPTOMATOLOGY. Since, as has been said, distinct obstruction in the trachea or bronchi occurred in all but a few of the cases (in all about 85 per cent.), the symptoms, for the most part, have been those of tracheal or bronchial stenosis.

Gerhardt[14] long ago noted that the symptoms could be divided into three stages:

1. Stage of irritation.
2. Stage of permanent stenosis.
3. Stage of suffocative attacks.

These three stages can, without doubt, be recognized in many of the cases, although the symptoms of the first stage have often been so slight as to pass unnoticed or at least unrecorded.

EARLIEST SYMPTOMS. The frequency with which the early irritative stage exists may be seen from an analysis of the cases with regard to the first symptoms noticed.

Among 69 cases of the present series in which that point could be determined, *cough* was the earliest symptom in 41. That is to say, in 60 per cent. of the cases a stage of irritation, as evidenced by the cough, was observed before any symptoms of obstruction appeared.

In 9 cases cough and dyspnœa together were given as the first symptoms. Twelve times dyspnœa alone was the first indication of trouble.

In a few remaining cases the first symptoms varied greatly. In 2 (II. and L.) the condition was latent until sudden, fatal hemorrhage, from ulceration and erosion of a bloodvessel took place. In one instance (LVIII.) the first and prominent symptoms were pain and tenderness referred to the upper part of the trachea. In 1 (XXX.) aphonia and dysphagia from laryngeal ulceration are mentioned as the first symptoms. In 1 (III.) there was a sudden suffocative attack requiring tracheotomy.

Finally, in Case X. attention was first called to the condition by friends of the patient who noticed a stridulous breathing sound.

DURATION OF SYMPTOMS. In the 58 cases in which the records were satisfactory upon this point the duration of the symptoms was as follows:

Over one year	14	cases, or	24 per cent.
Between six months and one year	12	"	21 "
Between one year and six months	26	"	45 "
Under one month	6	"	10 "

In 1 case (LXII.) the symptoms extended over a period of three years. In several instances they lasted for two years or more.

INDIVIDUAL SYMPTOMS. *Cough.* Among the 77 cases of this series in which the symptoms were recorded, cough was noted in all but 12 cases. It was therefore present in 84 per cent. of the cases at least.

Usually, as has been said, it appeared as the first symptom, and in many cases it remained a prominent one throughout the disease.

In 33 cases the character of the cough was not mentioned. In six instances it was described as dry; in twelve, as accompanied by expectoration. In 9 other cases the cough was at first dry and later showed expectoration. Once it was described as worse at night; once as paroxysmal; five times as harsh and distressing, and once (LXXIV.) as resembling the cough of thoracic aneurism.

Sputum. This is mentioned specifically in only 30 of the 77 cases (40 per cent.), although doubtless it occurred more frequently. It is described variously as mucous, mucopurulent, purulent, fetid, blood-stained, nummular, etc. Blood-stained sputa were seen in 8 cases. In Case XL., which completely recovered, both connective tissue and elastic fibres were found in the sputa. In 1 case (IV.) the patient expectorated fragments of the tracheal cartilages at intervals for two and a half years. In one instance (LXXVI.) one of the chief complaints was the inability to expectorate.

Hemorrhage. In addition to the 8 cases in which were seen blood-tinged sputa, free hemorrhage from the trachea or bronchi occurred in 6 of the present list of cases, and at least twice among those of Vierling's series. In 6 of these 8 cases the hemorrhage occurred from some large vessel and was immediately fatal. Thus:

Case II. Ulcer of left bronchus, with perforation of branch of bronchial artery.

Case XXV. Ulcer of lower part of trachea, with perforation of arch of aorta.

Case L. Ulcer of lower part of trachea, with perforation of superior vena cava.

Case LXIII. Signs of tracheo-bronchial syphilis. Fatal hemorrhage. No autopsy.

Case V., 32. Ulcer of right bronchus, with perforation of branch of pulmonary artery.

Case V., 42. Ulcer of right bronchus, with perforation of branch of pulmonary artery.

Dyspnœa This seems to have been the most constant, as well as the most conspicuous, of all the symptoms. In only 2 of the 77 cases (XXXVI. and LV.) is there a statement that it was *not* present. In five instances no mention of it is made. In all the other 70 cases (90 per cent.) it was present at some time.

Usually this difficulty of breathing appeared early (in 12 cases it was the first symptom), increased steadily, and occupied the foreground in a clinical picture as alarming and distressing as can well be imagined.

Concerning the type of dyspnœa (whether chiefly inspiratory or expiratory) the reports are very unsatisfactory. Whenever references

are made to this point, it is in connection with a description with the stridor present. The matter will, therefore, be discussed under that heading.

The dyspnœa presented itself in two distinct forms—*constant* and *paroxysmal.*

1. *Constant Dyspnœa.* Continuous, progressive dyspnœa, as distinguished from distinct paroxysmal attacks, was present in all but 2 of the 70 cases. In these 2 the disease showed itself in the form of sudden and severe paroxysmal dyspnœa. In all the other cases the onset was more or less insidious—a gradually developing breathlessness, at first, perhaps, only upon some exertion, but sooner or later becoming continuous and progressively worse.

This second stage of Gerhardt—that of permanent stenosis—varied in length between a few weeks and two or three years.

2. *Paroxysmal Dyspnœa.* At some time in the course of the disease, in *half* of the 70 cases with dyspnœa, there were added paroxysmal attacks of the most alarming air hunger, with orthopnœa, cyanosis, stridor, inspiratory sinking in of the tissues of the neck and epigastrium, rapid, feeble pulse, and sometimes even unconsciousness.

In 2 cases (III. and XII.) these suffocative attacks developed suddenly without any preceding constant dyspnœa. In all the other cases they appeared only after the other symptoms of dyspnœa and cough had been present some time.

Gerhardt's *third* stage, therefore, could be seen in 44 per cent. (34 out of 77) of all the cases. These attacks in some instances were of almost daily occurrence. In others they appeared only at long intervals. They were excited usually by some special exertion, such as climbing stairs, by attacks of coughing, or even by eating (LXXIX.). In 5 cases the attacks were especially prone to develop at night.

The suddenness and severity of some of these suffocative attacks seem wellnigh incredible. In Case XI., for example, the first symptom was an attack which followed the exertion of running quickly up a flight of stairs. This was followed by many other paroxysms of a similar character in which the patient would be for many hours unconscious, pulseless, and with barely perceptible respiration. Between these attacks she would be quite well.

In no less than 11 cases did the patient die in such a suffocative attack.

That permanent, organic stenosis of the trachea or main bronchi is sometimes accompanied by dyspnœa altogether or chiefly paroxysmal and intermittent has long been recognized, and was emphasized especially by Worms[19] in the report of his case (X.).

The explanation of this curious fact requires a consideration of the effects in general produced by permanent narrowing of the larger

tubes. Such studies of the influence upon respiration of a narrowing of the trachea have been made by different observers from both experimental and clinical standpoints, and the matter is discussed at length by Grossmann[20] in his admirable article on tracheal stenosis.

Grossmann calls attention to the fact that dyspnœa of two distinct sorts can be recognized; first, that with slow, abnormally deep breathing, and, second, that in which the respirations are rapid and shallow. In the first type the equilibrium is restored by the *depth*, in the second by the *frequency* of the respirations.

It has been found, both clinically and experimentally, that in tracheal stenosis there is, for a time at least, slowing and deepening of the respirations, slowing of the pulse rate, and an increase in the blood pressure in both the systemic and the pulmonary circulations.

The result of this is that the work of the heart is much increased. So long as the heart, by greater activity and hypertrophy, can fully meet this increased demand upon it, compensation is maintained and the subjective evidences of dyspnœa may be lacking altogether.

It is to the failure of such compensation by the heart that Grossmann attributes all the disturbances in the further course of tracheal stenosis. He compares the effect upon the heart in such conditions to that produced by bodily overexertion—*e. g.*, in "soldier's heart," etc.—and says that the clinical picture and course of the disease are the same in both cases—*i. e.*, that of cardiac insufficiency.

The *post-mortem* findings, also, he thinks, give support to this view—passive congestion of the liver, spleen, and kidneys, together with dilatation of the heart (and in particular of the right ventricle), and a marked engorgement of the lungs.

This engorgement of the lungs seems to take part in the production of further dyspnœa in a very interesting way.

The distention of the alveolar capillaries from the engorgement is believed to produce a rigidity of the lungs which seriously interferes with their respiratory expansion and contraction. Von Basch,[22] who advocates this idea, found that in animals engorgement of the pulmonary capillaries could interfere with the expansion of the lungs to such an extent that the difference between inspiration and expiration could scarcely be appreciated.

In this way is the vicious circle made more complete, and to the tracheal dyspnœa added the more serious, progressive cardiac dyspnœa. The slow, deep respirations are replaced gradually by rapid and shallow ones, the pulse becomes rapid and feeble, and the picture then becomes that of grave cardiac insufficiency in which, however, certain evidences of the primary stenosis (stridor, etc.) are usually still to be recognized.

To return now to the cases where intermittent, paroxysmal dyspnœa is the conspicuous symptom of permanent stricture of trachea or

bronchi. It is the usual history of such cases, as has been said, that the attacks of dyspnœa follow directly upon some special exertion, such as stair climbing, attacks of coughing, etc. If we imagine in such cases that, by reason of deeper and slower respirations and more vigorous heart action, respiratory and cardiac compensation has been maintained, but only just maintained, in spite of the stricture, it is easy to see how some comparatively slight exertion might add sufficient work to the already overworked heart to upset the delicately adjusted balance. Then to the slight, and, perhaps, altogether unnoticed, constant dyspnœa would be added the paroxysmal attack. The failure of cardiac compensation would be followed by failure of respiratory compensation which would show itself in the form of a suffocative attack. Under the absolute rest made necessary by such an attack it is also easily conceivable that the heart should gradually regain its equilibrium, and that the paroxysmal dyspnœa should be replaced by the slight, and purely tracheal, constant dyspnœa. In the same way any transient inflammatory swelling of the mucous membrane of the trachea and bronchi, which might temporarily still further diminish the lumen, would cause increased labor for the heart, and might, therefore, precipitate such an attack of paroxysmal dyspnœa.

Rate of Breathing. In the reports of cases of syphilis of the trachea and bronchi very little can be found concerning the actual rate of breathing. In most of the cases where the rate is definitely stated the condition of stenosis had existed for some time, and, to the purely tracheal dyspnœa, there had been added that due to cardiac insufficiency; so that the respiratory rate has usually been above the normal. In many of the cases, however, although the number of respirations to the minute is not mentioned, the comparative slowness of the breathing is implied by the statement that each inspiratory act was greatly prolonged. Finally, in a few cases the respiratory rate is given as distinctly below the normal. The most striking example of this is seen in the case of Worthington[5] (almost the first case reported), in which, owing to the great prolongation of the inspiration, the whole breathing act occupied a period of about ten seconds—the respirations averaging not more than six to the minute.

Stridor. Among all the symptoms of tracheo-bronchial syphilis none is perhaps so conspicuous and striking, when present, as the peculiar stridulous sound which, in so many of the cases of stenosis, accompanies one or both respiratory acts. Its occurrence among the 77 cases whose symptoms are here analyzed may be tabulated as follows:

Stridor (whether inspiratory or expiratory not stated)	17 cases.
" with both inspiration and expiration	8 "
" with inspiration alone	12 "
" with expiration alone	1 "

Stridor, then, was mentioned as present in about 50 per cent. of the cases. As the reports are, in some instances, decidedly meagre in the details of the symptoms, it is very probable that this figure is somewhat below that of its actual frequency.

Among the 21 cases in which the respiratory time of the stridor is definitely stated, it will be seen that in 12 the sound occurred with inspiration alone, and in 8 only, with both inspiration and expiration. In most of these latter cases, moreover, the stridor is spoken of as having been more intense with inspiration than with expiration. No instance is given in which this rule was reversed. In some cases the sound has been only inspiratory during quiet breathing, but upon any exertion has been audible during expiration as well.

In only *one* of the cases was the stridulous sound present with expiration alone. In this case (XI.), which in some respects is unique, the dyspnœa as well as the stridor was almost altogether with expiration, and was due chiefly to the valve-like action of a large piece of eroded cartilage which, with expiration, would be lifted up and would almost completely close the tracheal lumen.

It may safely be concluded, I think, from all this, and from the fact that the marked prolongation of inspiration is commented upon in so many of the reported cases, that with syphilitic stricture (as with other forms) the dyspnœa and the stridor are regularly most distinct with inspiration, and that very commonly they are evident only during that part of the breathing act.

It is evident that the intensity of the stridor will depend not alone upon the size of the stricture, but, as well, upon the length and vigor of the respirations, and that, *cæteris paribus*, the deeper and more powerful the inspiration the more pronounced is likely to be the accompanying sound. If this be the case we should expect the stridor to be most marked in the cases where the dyspnœa due to the stenosis had not yet been augmented by that due to a failing heart; in other words, where the respirations were still slow and deep, rather than rapid and superficial.

A study of the cases seems to support this assumption, although the lack of detail in many of the histories makes it difficult to reach a definite conclusion.

It is certain that not every case of stricture is accompanied by stridor. In Case LI. the symptoms were those only of emphysema; there was no stridor whatever, and yet the autopsy showed, in addition to emphysema, a considerable stricture of the trachea and great narrowing of the left bronchus. In Case LVI. the symptoms were entirely pulmonary, no evidence of tracheal stenosis being present, but the autopsy revealed many gummatous swellings and bands of scar tissue throughout the trachea.

In Case XX. the clinical picture was that of chronic pulmonary phthisis, although both bronchi were shown, *post-mortem*, to have been greatly contracted. In Case LVIII. a gummatous tumor in the upper part of the trachea, which almost filled its lumen, gave rise to no stridor.

On the other hand, in Case LXIV., Gairdner observed, in addition to slight, constant dyspnœa and cough, attacks of paroxysmal dyspnœa, accompanied by inspiratory stridor. At the autopsy there was found a very large ulcer involving almost the whole circumference of the lower end of the trachea and extending into the main bronchi, but no mention whatever is made, however, of any stenosis of either trachea or bronchi.

Concerning the *character of the stridulous sound*, the descriptions differ widely. Its most characteristic form seems to be that which was first so graphically described by Worthington,[5] who compares it to the "roaring" sound made by unsound horses. The French writers, in their term *cornage*, have adopted the same comparison. But the tone is by no means always "roaring" in character. Sometimes the pitch is much higher and the sound is whistling or sibilant. In some cases the writers have described it as exactly similar to the stridor produced in the larynx by paralysis of both posterior crico-arytenoids (LIX.). Indeed, in most cases, the sounds seem to be indistinguishable from those produced in laryngeal stenosis.

The *intensity* of the stridor varies greatly as might be expected. In several cases it is described as so loud as to interfere with the sleep of other patients in the ward. Sometimes it has been a faint sibilant sound scarcely audible except upon exertion. In two cases (XXXVII. and LIX.) it was especially loud during sleep.

The stridor, moreover, does not seem to depend upon the location of the stricture. It is heard when the bronchi alone are stenosed, and may be loud even when only one bronchus is narrowed (X.).

Pain. Pain of some sort is mentioned in 13 reports (17 per cent.). In 3 cases (VII., X., and XV.) it was felt as a dull, constricting feeling behind the sternum. In Case VI. pain was felt at the level of the larynx, although the lesions were located at the lower end of the trachea and in the bronchi. Twice (XXI. and LXVIII.) was the pain referred to the suprasternal notch. In the first case there was only a cicatricial stricture at the bifurcation; in the second there was extensive ulceration of the trachea between the sixth and ninth rings, as well as stricture of the trachea at the bifurcation and of the right bronchus.

In 4 cases (XXXVI., XXXIX., XLII., and LXX.) the location of the pain is not given other than as in the throat or trachea. Gnawing pain was felt between the shoulders in Case LV., in which the autopsy showed many small ulcers throughout the trachea and bronchi.

In Case LVIII. pain and tenderness just below the larynx were the conspicuous symptoms, and were due to a gumma situated in the trachea at that point. Pain and tenderness were caused at about the same point in Case LXVI. by compression from a peritracheal mass.

It seems doubtful, from a study of the cases just mentioned, if the location of the pain is of much value in determining the position of the lesion.

Tenderness. Tenderness on pressure is mentioned only three times in all. Once it was noticed over the larynx in a case (XXXIX.) in which the larynx was involved in the lesion; once there was great tenderness over the upper part of the trachea, with a gummatous tumor on the inner surface at that point (LVIII.), and once (LXVI.) over the right side of the trachea in a case with extensive peritracheal induration.

Voice. Reference to the voice was made in 34 of the cases. Among these it was found normal in 10 (30 per cent.). In 4 other cases it was clear but weak. Twice (XXXIII. and XXXV.) there was complete aphonia, although the larynx was normal. In both of these cases there was marked stenosis from membranous bands situated just below the larynx.

Aphonia occurred in 2 other cases, but in each of these there was some lesion of the larynx. In 6 cases (IX., XXXVIII., XLIV., LXVI., LXXIV., and LXXXII.) the larynx was found normal, although the voice was hoarse or rough. In another case, with a normal larynx (XXXVII.), the character of the voice varied greatly; at times there would be aphonia, at other times the voice would be harsh and rough, at still others it would be quite clear.

It is evident, therefore, in tracheal and bronchial syphilis, that, even with a normal larynx, the vocal sounds may assume almost any abnormal character and may even disappear altogether, and that, therefore, a hoarse or even whispering voice cannot be taken as evidence that the obstruction is in the larynx rather than below.

Retraction of Tissues during Inspiration (*Tirage*). A distinct sinking-in of the tissues during inspiration was observed in 9 of the 77 cases. In most of these this was apparent not only at the root of the neck, but also in the epigastrium and along the free border of the ribs.

Movement of Larynx. Gerhardt,[14] in his classical description of the signs of tracheal stenosis, lays much weight upon the limitation of vertical movement of the larynx in this condition as compared with that of laryngeal stenosis, and goes so far as to say "in spasmodic and stridulous breathing laryngeal movement of less than one centimetre is a certain sign of tracheal or tracheo-bronchial stenosis."

Statements concerning the mobility of the larynx can be found in only 6 of this series of cases. In 2 (XXX. and XXXVII.) the larynx

is described as not moving at all with respiration; three times (XLVII., LII., and LXXVI.) it is said to have moved only very slightly, and in 1 case (XXXVIII.) the amount of respiratory excursion is given as one centimetre. These few statements, therefore, as far as they go, support the view of Gerhardt, since in only 1 of the 6 cases was there anything more than very slight movement, and in this case the movement (1 centimetre) was considerably less than one is apt to see in conditions where the obstruction is in the larynx.

Pulmonary Signs. Physical examination of the lungs in the different cases gave results of the most miscellaneous character. In 12 of the cases the respiratory murmur was entirely masked by the loud stridor. In another instance it was obscured by tracheal rhonchi.

In 10 cases the signs are described as those of bronchitis. In two others (XX. and XLIX.) the pulmonary signs, as well as the clinical features in general, were those of chronic phthisis. In Case LXXIII., with stricture of the left bronchus, there was loud tubular breathing over the right chest, while the breathing was inaudible on the left side.

In a number of the cases the respiratory sounds were very feeble over both chests. Among those cases which were complicated by pneumonia there were, of course, corresponding signs. In many instances no careful description of the physical signs of the lungs is given.

Miscellaneous Symptoms. In a number of the cases (XX., XXXV., XXXVIII., XLII., XLIX., LV., LVI., LXVI., and LXXXI.) emaciation was a conspicuous feature. In some of these cases there were profuse sweats as well, and these, with the emaciation and cough, made the resemblance to chronic phthisis very strong. In several instances (especially in XXIII. and XXXVIII.) attacks of chills and fever were common.

Dysphagia was noticed three times (XVIII., XXXIX., and LXX.). In one of these there were lesions of the larynx as well as of the trachea. In Case LVII. a communication between the trachea and œsophagus was formed by ulceration, with the subsequent entry of food into the trachea during swallowing, and, eventually, death from inhalation pneumonia.

A *pulsus paradoxus* was noted in 1 case (LXXXI.), with the signs of tracheal stricture.

In case LXXVI., in which both inspiration and expiration were very labored, a soft, tumor-like prominence appeared above the left clavicle, which was tympanitic on percussion and which was regarded as an emphysematous ballooning of the apex of the left lung.

Another curious and rare condition was that of tracheocele (LXXV.). The patient, a man, aged twenty-five years, noticed the gradual development of a tumor in the median line of the neck just below the larynx,

which in six weeks reached the size of a walnut. Laryngoscopical examination showed, in addition to evidences of syphilis in the throat, an irregular ulcer on the anterior wall of the trachea. Pressure upon the tumor in the neck would cause it to diminish in size, a hissing sound could be heard in the throat, and air and fluid could be seen to enter the trachea through the ulcer.

Diagnosis. For the purposes of diagnosis, cases of syphilis of the trachea and bronchi may be conveniently divided into: 1. Those without signs of obstruction to breathing. 2. Those with such obstruction.

In the first class of cases the recognition of the nature of the trouble will usually be a very difficult and often a quite impossible, task. The symptoms likely to be present—cough, pain, blood-stained sputa or free hemorrhage—are all of them common to various pulmonary conditions of much more frequent occurrence, and the suspicion of tracheobronchial syphilis could hardly be raised except by the discovery in the trachea, by laryngoscopical examination, of ulcers or localized swelling, or by the presence of such a group of symptoms in patients showing other syphilitic manifestations, and in whom the other more common affections could with reasonable certainty be excluded.

In the second class, which, fortunately for diagnosis, includes most of the cases, the question resolves itself into two parts: 1. Does tracheal or bronchial obstruction exist? 2. Is such obstruction due to syphilis?

Obstruction in the larger air passages—*i. e.*, larynx, trachea and large bronchi—manifests itself in a clinical picture which is usually characteristic and unmistakable. This consists of: 1. A peculiar type of dyspnœic breathing, in which the prolonged, labored, and relatively slow inspiration and the shorter, easy expiration follow each other without the usual pauses. 2. A stridulous sound chiefly or altogether inspiratory. 3. In most cases an inspiratory sinking-in (*tirage*) of the tissues of the root of the neck, the epigastrium, and the lower intercostal spaces.

The differentiation of laryngeal obstruction from that occurring in the trachea or bronchi should present no difficulties except in cases where it is not possible to make a satisfactory laryngeal examination. These may be met in children and also in adults whose throats are intolerable to the necessary manipulations, or whose dyspnœa is so intense as to make such an examination impossible.

Even without an inspection of the larynx the differentiation can usually be made, although it must be admitted that the difficulties to be met may be very great. Careful examination of the throat, at least, can be made in all cases, with the view of excluding such obstructive conditions as retropharyngeal abscess and quinsy.

The points likely to be helpful in distinguishing tracheo-bronchial stenosis from that occurring in the larynx may be enumerated as follows:

1. History.
2. Character of voice.
3. Amount of respiratory movement of the larynx.
4. Attitude of the head during inspiration.
5. Location of the inspiratory thrill.
6. Point of greatest intensity of stridor.
7. Variation in the signs over the two sides of the chest.

Careful inquiry into the history of the case, concerning mode of onset, character of cough, position of pain, dysphagia, hoarseness, etc., is likely to reveal some fact of value in determining the position of the obstruction.

The character of the voice is of much less diagnostic value than might at first be supposed. As has already been shown, the voice is frequently hoarse, and may be even aphonic, in cases of tracheal stenosis with normal larynges. On the other hand, in one condition of laryngeal obstruction—that of bilateral paralysis of the abductor muscles—the voice may be quite unaffected.

The typical voice of tracheal stenosis is a light, clear one, of somewhat restricted range, and in conditions of obstruction of the large air passages the presence of such a voice would suggest that the stenosis existed at some point below the larynx. It should be borne in mind, however, that such a voice would be quite compatible with at least one form of laryngeal obstruction.

It was Gerhardt[14] who first emphasized the fact that in laryngeal stenosis the normal slight downward movement of the larynx during inspiration was much increased by the impact of the inspired air against the upper surface of the obstructed glottis, and that in an effort to offset this and to afford a point of support to the larynx the head was retracted and the chin elevated. He asserted that in dyspnœa due to obstruction *below* the larynx this exaggerated movement of the larynx did not occur, nor was the head retracted, and he believed, therefore, that these two points furnished valuable aid in differentiating from laryngeal stenosis that of the trachea and bronchi.

Among the cases here analyzed (as has been said under Symptomatology), Gerhardt's assertion that in tracheal stenosis the larynx shows little or no respiratory movement, seems to be borne out by the records of the few cases in which the point is mentioned.

The other half of the dictum, however (that in laryngeal stenosis there is always an exaggeration of the normal respiratory movement of the larynx), rests, I think, upon a less firm basis. There is reason to believe that, occasionally at least, in laryngeal obstruction the elevation of the chin and the contraction of the muscles supporting the

larynx may be so great as not only to prevent any exaggeration of the normal downward excursion, but even to cause a slight inspiratory elevation of that organ.

Gerhardt's conclusion, therefore, that in labored and stridulous breathing a laryngeal movement of less than one centimetre indicates always tracheal or tracheo-bronchial stenosis, must be accepted with some reserve. Where the excursion is greater than one centimetre, however, there is strong presumptive evidence that the obstruction is in the larynx rather than below.

The attitude of the head and chin, likewise, seems to be not altogether trustworthy. At least Neumann[23] reports a case of tracheal stenosis, in a child, due to enlarged glands in the thorax, in which the head was retracted and the chin elevated throughout.

Careful palpation of the larynx and trachea will sometimes lead to the localization of the obstruction by the finding of the point at which an inspiratory thrill can be most distinctly felt. Such a thrill, however, is by no means always present.

In the same way the point at which the stridor is loudest may sometimes be readily determined by the stethoscope, and this will usually correspond to the point of obstruction. Where the stricture lies behind the sternum, however, the stridor might, as Gerhardt points out, be heard louder over the larynx than over the point of stricture. In this connection it should be mentioned that Aufrecht[24] noticed in 3 cases of stenosis of the lower part of the trachea that the normal tracheal breathing sound could not be heard in listening over the trachea with the stethoscope, and he believes this disappearance of the tracheal breathing sound to be a sign of some value in tracheal stenosis.

No difficulty should be met in distinguishing laryngeal stenosis from that of a single bronchus. The diagnosis of the latter condition can usually readily be made by a careful comparison of the signs of the two sides of the chest. On the affected side there will be found usually restricted expansion, diminished vocal fremitus, enfeebled or absent respiratory sounds, and diminished vocal resonance.

It having been determined that the stenosis is in the trachea or bronchi rather than in the larynx, there remains the often more difficult task of deciding upon the nature of the obstruction. Stenoses due to syphilis must be distinguished from those due to a great variety of other causes, some of which are of much more frequent occurrence.

These non-syphilitic forms of tracheo-bronchial stenosis may be conveniently grouped into (1) those due to compression from without, and (2) those caused by obstruction from within.

The conditions likely to cause compression of the trachea along its course in the neck, such as goitre, enlarged lymph nodes, etc., can usually be readily recognized. In the thorax, however, such compres-

sion of the trachea or bronchi may be very difficult to demonstrate. The list of causes includes thoracic aneurism, enlargement of the tracheo-bronchial and mediastinal glands in tuberculosis, pseudo-leukæmia, etc., tumors and abscess of the mediastinum, carcinoma of the œsophagus, enlargement of the thymus gland, etc. It must be remembered in this connection, moreover, that syphilitic enlargement of the tracheo-bronchial lymph nodes may be added to the lesions of the interior of the trachea or bronchi.

Of the possible causes of obstruction operating from within there are to be mentioned:

1. Foreign bodies.
2. Simple and malignant new-growths (polypi, primary carcinomata).
3. Lupus.
4. Chronic glanders.
5. The chronic blennorrhœa of Stoerck.
6. Tuberculosis.
7. Leprosy.
8. Cicatrization of the ulcers of variola.
9. Scars following the use of intubation or tracheotomy tubes.
10. Hysterical spasm.
11. Accumulations of mucus, etc.

The diagnosis of syphilitic stenosis from obstruction due to a foreign body would, for obvious reasons, hardly offer difficulty.

Mucous polyps and primary carcinomata of the trachea, while exceedingly rare, do occur and might be impossible of differentiation from syphilitic disease unless seen by the laryngoscope. A history of long-standing cough, of blood-stained sputa, of tracheal pain or tenderness preceding the signs of obstruction, together with a history of syphilis and especially of severe, late manifestations, would point toward the latter disease as the cause of the stenosis.

Regarding the appearance of tuberculous disease in the trachea, Wright[35] says that he can find the record of only a single case in which tuberculosis was either primary in the trachea or was confined to it to the exclusion of the larynx. A case of spasmodic stricture of the trachea, ascribed to hysteria, has been reported by Landgraf.[36] An instance of sudden bronchial obstruction due to the accumulation of thick mucus during an attack of acute bronchial catarrh is recorded by Deri.[37]

Prognosis. *Mortality.* In Vierling's list[15] of 46 cases death occurred in 39; 3 were cured, and more or less improvement was seen in the remaining 4. In the present series of 82 cases there were 58 deaths and 15 cures, while the remaining 9 were reported as improved.

The mortality among the 128 cases, therefore, was 76 per cent. The

number of cures is given as 18 (14 per cent.), while in 10 per cent. improvement of greater or less degree was recorded.

Cause of Death. Among the 58 fatal cases in the present series, death was caused in 19 by the development of some form of pneumonia. In 2 of these cases the pneumonia was the result of the inhalation of food particles after perforation of the œsophagus.

In 11 of the cases death was directly produced by a paroxysmal suffocative attack. In 4 cases it was due to sudden profuse hemorrhage from perforation of one of the large bloodvessels.

In most of the remaining cases the fatal ending came as the result of exhaustion and gradual cardiac failure. In a few instances it was due to syphilitic lesions in other parts of the body or to some intercurrent disease.

The relation of prognosis to the *early recognition of the nature* of the affection is clearly seen among the cases of this series. Of the 15 cures 13 occurred in cases where the lesions (gummata or ulcers) could be seen by the laryngoscope, and were thus early recognized and treated. In a fourteenth case, although the laryngoscopical examination failed, the diagnosis was readily made from the presence of syphilitic manifestations in other parts of the body. In only 1 case (LXXVI.) was a cure effected where the lesions were of long standing, and where the obstruction was due to cicatricial contraction.

The prognosis bears a very definite relation also to the *position* of the lesions. It is evident that in general the higher in the trachea the lesions the more readily, and, therefore, the earlier, will the diagnosis be made. So also when the disease is high up in the trachea are the cases much more susceptible of successful treatment by mechanical measures.

From what has been said it is obvious that the *character* of the lesions also will greatly affect the prognosis. Gummata and ulcers, in most cases, readily respond to antisyphilitic treatment. When the obstruction is due to contracting bands of cicatricial tissue much less is to be expected from treatment by drugs.

The condition of the other organs, especially of the lungs and heart, must greatly influence the duration of life and the chances of relief. This subject has been discussed at some length under the head of Dyspnœa. So long as the respiratory acts, in cases of obstruction, are slower and deeper than normal, it is safe to assume that no serious secondary changes have yet taken place in the lungs or heart. Such pulmonary and cardiac insufficiency is usually promptly shown by a change in the character of the dyspnœa by which the breathing becomes rapid and superficial and the characteristic picture of stenotic dyspnœa is replaced to some extent by the more familiar one of cardiac dyspnœa.

TREATMENT. The results of treatment have been studied in the 82 cases which make up the present series. In only 29 of these was a rational treatment carried out sufficiently to warrant the formation of a judgment as to its efficacy. In all the other cases either the nature of the condition was not recognized or the patient was seen only after some grave complication had developed and had rendered the case hopeless.

Among these 29 cases only 3 failed to respond satisfactorily to treatment. One of these was a case of inherited syphilis in which the character of the lesion was not determined (XVIII.). One (LXXIII.) showed diffuse fibrous thickening of the left bronchus with great stenosis, and in one (LXVI.) the obstruction was due to compression from extensive peritracheal deposit (in this case treatment was begun only five days before death).

Of the other 26 cases 15 were cured and 11 showed improvement of greater or less degree. In most of the latter cases, moreover, the improvement was marked and apparently permanent.

Among the 15 cases cured the character of the lesions in the trachea could be made out in all but 1. These 14 cases included 9 (XIV., XIX., XXXVII., XLI., LVIII., LIX., LXXII., LXXIV., and LXXX.) in which the symptoms were due to circumscribed or diffuse gummata; 3 (XXVII., XXXVI., and LXXV.) to ulceration; 1 (XXXIII.) to membranous bands forming a sort of false glottis, and 1 (LXXVI.) to cicatricial stenosis of the lower end of the trachea and of the left bronchus.

The nature of the lesion in the 11 cases improved by treatment could be determined in only 4. In 3 of these (XXX., XLV., and LXXVIII.) there was a gummatous thickening of the tracheal wall. In 1 (XLVI.) a diaphragm-like membrane existed at the third tracheal ring. The very few cases of cicatricial stenosis in which relief was obtained were all (except Case XXXIII.) benefited only by mechanical measures.

As regards the details of anti-syphilitic treatment, the greatest benefit seems to have been obtained from large doses of the iodides, although some of the cures were effected by the combination of mercury and potassium iodide. Some of the early French writers (*e. g.*, Vidal[28]) believed that too rapid cicatrization of the ulcers or gummata tended to increase the dangers of subsequent stenosis, and they took pains, therefore, to interrupt the treatment from time to time in the hope of avoiding this complication. By most of the later writers this precaution is regarded as unnecessary, to say the least, and the drugs have usually been pushed to the limit of tolerance. In 1 case (LXIV.) Ingals saw little result from 30-grain doses of sodium iodide, but when the drug was increased to 120 grains t. i. d. there was marked improvement.

Such large doses of the iodides, however, are not altogether without danger because of the sudden œdema of the mucous membrane which occasionally develops. Wright[28] and others have seen instances of alarming dyspnœa produced thus in cases of tracheal and laryngeal stenosis.

McKenzie[29] believes that cases of syphilis of the air passages have been benefited and cured by the occurrence in the patient of some acute febrile disease such as scarlatina, measles, or erysipelas. This idea is indorsed by Parrain.[30]

In addition to the treatment by antisyphilitic remedies, satisfactory and even brilliant results have been obtained in a few cases of stenosis by mechanical means. These measures consist in: 1. The gradual dilatation of a stricture by the introduction of catheters through the larynx. 2. Such dilatation through a tracheotomy wound. 3. The use of a tracheotomy canula.

Relief was obtained by the introduction of sounds through the larynx in 3 cases. This successful dilatation of a tracheal stricture by catheterization was first demonstrated by Schroetter[31] in a case of cicatricial stenosis following syphilitic ulceration (XXVII.). The scar of the healed ulcer formed a membranous constriction of extreme grade, which was gradually dilated by catheters and hard rubber bougies of increasing size. The patient eventually learned to introduce the catheter himself (see further account by Arnold[32]), and was sent home entirely relieved, with instructions to continue the daily introduction of the catheter. (In a case also of cicatricial narrowing of the entrance of both main bronchi, believed to be due to variola, Schroetter[33] obtained considerable relief from the same method of treatment.)

The second case of syphilitic stenosis relieved in this way was that of Massèi[34] (XLVI.). A membranous stricture, at the level of the third tracheal ring, admitting only a No. 12 French catheter, was greatly improved by gradual dilatation.

The third case of syphilitic stricture relieved thus was reported in 1895 by Seifert.[35] The results reached in this case are so remarkable as to warrant its citation in some detail.

A man, aged forty-three years, was sent to Gerhardt, with the diagnosis of tracheo-bronchial stenosis from gummatous cicatrization. There had been a gradually increasing difficulty of breathing for one year, shown especially upon any exertion. Antisyphilitic treatment had given only slight relief. Examination showed much slowing of the respiration, with lengthening of both acts, a loud inspiratory and expiratory stridor, inspiratory sinking-in of the epigastrium, almost complete cessation of respiratory activity over the left lower lobe, and a tumor-like bulging above the left clavicle, giving a tympanitic note on percussion. The larynx moved only slightly with the labored respirations, and the

laryngoscope showed it to be normal. In the trachea slight stenosis could be noticed at the third cartilage, and a more marked narrowing immediately above the bifurcation. A diagnosis could be made of cicatricial stenosis of the trachea and left main bronchus. The prominence above the left clavicle was believed to be an emphysematous ballooning of the left upper lobe. Seifert first dilated the tracheal stricture by means of Schroetter's bougies and obtained some relief from this. He then essayed the catheterization of the left bronchus. Beginning with a bougie of 5.5 mm. he was able gradually to introduce larger and larger sounds until he could enter the main bronchus with one of 12.5 mm. diameter. After ten weeks of almost daily catheterization the patient was discharged in excellent condition, with the disappearance of almost all of the symptoms, including the emphysematous bulging above the clavicle. The subsequent history of the case is not given.

The successful catheterization of a bronchus was first reported in 1887 by Landgraf,[35] who obtained considerable temporary relief in a case of stenosis of the left bronchus, which was thought at the time to depend upon cicatricial contraction, but which eventually proved to be due to the pressure of an aortic aneurism!

There is no doubt that the trachea and bronchi can acquire a surprising degree of tolerance to such instrumentation. Indeed, in the cases cited the reaction following the frequent passage of bougies was more apparent in the larynx than in the parts below.

Tracheotomy. The record of the performance of this procedure among the cases of tracheo-bronchial syphilis here cited is instructive in that it shows how futile and irrational, in many cases, was the operation, and emphasizes anew the great importance of an accurate diagnosis of both the character and the seat of the lesion.

Tracheotomy was performed seventeen times. In 10 cases (V., VI., XXI., XXIV., XXXII., LII., LX., LXV., LXX., and LXXI.) no good whatever was accomplished by the procedure, the obstruction being situated entirely or in great part below the level of the canula. In three cases (III., XVI., and XXXIV.) slight and temporary relief was noticed, although it was found later that the tube did not reach the point of obstruction.

Two other cases, in which the dyspnœa was due partly to tracheal stenosis and partly to paralysis of the abductors of the larynx, showed considerable improvement which was, however, only temporary.

Finally, in 2 of the 17 cases tracheotomy was of real and permanent value. In 1 of these (XV.) an examination with the finger through the wound revealed the stricture some distance below. By means of a long tube this obstruction was passed and the symptoms were relieved. Nineteen months later the patient was alive and was still wearing the

tube. (It would seem in this case as though much good might have been accomplished by gradual dilatation of the stricture through the tracheotomy wound.) In the second case (XLV.) the stricture was situated one-half inch below the larynx and followed gummatous deposit at that point. The dyspnœa was relieved by tracheotomy below, and under large and interrupted doses of potassium iodide the obstruction was so much improved that after six months the tracheotomy tube could be discarded.

The purposes and limitations of tracheotomy should be clearly kept in mind. It cannot be gainsaid that in the past the operation has often been done hastily and ill-advisedly. In tracheo-bronchial syphilis it can serve only one of two purposes: first, to relieve urgent dyspnœa by admitting air to the trachea below the point of obstruction, and, second, to aid in the mechanical treatment of stricture by the introduction of bougies or canulas. Concerning the first use it is evident that this must be limited to lesions of the upper end of the trachea. As a means of furnishing an entrance for instruments for the dilatation of strictures its value is somewhat uncertain. It may be doubted whether a stricture situated at the lower end of the trachea or in a bronchus can be approached any more easily through a tracheotomy wound than through the larynx. In either case the matter is difficult enough and demands the skill of an expert laryngologist. The latter method, moreover, has certain obvious advantages over tracheotomy in the diminished likelihood of a complicating inhalation pneumonia.

The following are the condensed records of 81 cases not hitherto collected, which, with my own case and the 46 tabulated by Vierling, form the basis of this paper:

Case I.—Munk.[37] Male, aged nineteen years. Syphilis one and one-half years before. Cough, purulent expectoration and emaciation for some months. Death. *Autopsy.* Many small ulcers in larynx and throughout all the bronchi.

Case II.—Watson.[7] Man. Evidences of late syphilis. Slight cough with sudden fatal hemorrhage. *Autopsy.* Large ulcer in left bronchus perforating branch of bronchial artery.

Case III.—Gillespie.[38] Man, syphilitic. Sudden suffocative attack. Tracheotomy. Death. *Autopsy.* Ulcers in trachea and both bronchi. Right bronchus constricted.

Case IV.—Jones.[39] Man, aged thirty-one years. Cough. Expectoration of fragments of tracheal cartilages. Death two and a half years later. *Autopsy.* Many scars in lower part of trachea and in both bronchi. Constriction, especially of left bronchus.

Case V.—Charnal.[40] Man, aged thirty-six years. Syphilis twelve years before. Cough and increasing dyspnœa for three months. Labored, stridulous breathing. Suffocative attack. Tracheotomy. Death. *Autopsy.* Cicatricial stenosis of lower part of trachea. Dilatation of bronchi.

CASE VI.—Boeckel.[41] Woman, aged fifty-six years. Evidences of late syphilis. Cough. Increasing dyspnœa, with suffocative attacks. Tracheotomy. Death. *Autopsy.* Cicatricial stricture at bifurcation, involving left bronchus. Old tuberculosis at apices of lungs.

CASE VII.—Vidal.[28] Woman, aged thirty-two years. Syphilis three years before. Cough. Emaciation. Increasing dyspnœa. Stridor. Normal larynx. Voice unchanged. Dull pain behind sternum. Under KI all symptoms disappeared, except stridor on exertion.

CASE VIII.—Steiner.[42] Boy, aged twelve years. Inherited syphilis. Cough. Dyspnœa. Emaciation. Stridor. Orthopnœa. Death after several months. *Autopsy.* Lower half of trachea much constricted by scars. Small polypoid tumor above bifurcation. Right bronchus much stenosed. Enlarged bronchial glands. Perihepatitis.

CASE IX.—Lancereaux.[43] Woman. For three weeks hoarse voice, cough, and gradually increasing dyspnœa. Orthopnœa. Death from erysipelas. *Autopsy.* Stenosis of lower part of trachea and of left bronchus. Ulceration of trachea and right bronchus. Gummata of liver.

CASE X.—Worms.[19] Woman, aged thirty-four years. Syphilis five years before. For six months a stridulous sound with breathing. Increasing dyspnœa. Cough. Attacks of suffocation. Sudden death. *Autopsy.* Enormous cicatricial stricture of left bronchus, with disappearance of cartilages. Enlarged bronchial glands.

CASE XI.—Prengrueber.[44] Girl, aged eighteen years. Chancre nine months before. Increasing dyspnœa. Violent suffocative attacks. Expiration, especially labored, prolonged, and stridulous. Death. *Autopsy.* Extensive ulceration of trachea with necrosis of cartilages. Valve-like action of detached cartilage causing obstruction during expiration.

CASE XII.—Morell Mackenzie.[45] Man, aged thirty-nine years. Sudden development of attacks of intense dyspnœa. Well between attacks. Ultimate death. *Autopsy.* Cicatricial stenosis of lower part of trachea. Lobular pneumonia. Gummata in liver and right kidney.

CASE XIII.—Erichsen.[46] Man, aged thirty-eight years. Syphilis for seventeen years. For three months cough, dysphagia, and dyspnœa, especially at night. Loud stridor. Hoarse voice. Improvement under anti-syphilitic treatment.

CASE XIV.—Zurhelle.[47] Young woman. For several weeks cough and increasing dyspnœa. Whole of visible trachea greatly narrowed. Swollen mucous membrane covered with exudate and showing superficial ulceration. Under KI recovery in six months.

CASE XV.—Rey.[48] (First case.) Woman, aged forty-two years. Evidences of late syphilis. For one month cough and increasing dyspnœa. Suffocative attack. Tracheotomy. Long canula passes stricture. Patient living and wearing canula nineteen months later.

CASE XVI.—Rey.[48] (Second case.) Man, aged forty-three years. Syphilis for ten years. Dyspnœa. Stridor. Suffocative attack. Tracheotomy. Death from pneumonia and empyema. *Autopsy.* Scars and ulcers throughout larynx and trachea.

CASE XVII.—Rey.[48] (Third case.) Man, aged thirty-nine years. Chancre nine years before. Cough. Increasing dyspnœa. Repeated suffocative attacks. Loud inspiratory stridor. Sudden death. *Autopsy.*

Stricture of trachea above bifurcation. Ulceration and dilatation above this.

CASE XVIII.—Rey.[48] (Fourth case.) Girl, aged two and a half years. Mother syphilitic. Symptoms appeared at age of ten months. Cough, hoarse voice, dyspnœa, and intense suffocative attacks. Stridor. No improvement by treatment.

CASE XIX.—Ives.[49] Man, aged thirty-five years. Syphilis fourteen years before. For six months cough, increasing dyspnœa, and stridulous breathing. Paralysis of left vocal cord. Gummatous mass seen in upper part of trachea. Complete recovery under mixed treatment.

CASE XX.—Pye-Smith.[50] Man, aged forty-two years. For two years all the symptoms of pulmonary tuberculosis; then cerebral symptoms and death. *Autopsy.* Both bronchi much contracted and misshapen. Extensive peribronchitis. Interstitial pneumonia. General dilatation of the bronchi. Gumma of dura mater.

CASE XXI.—Obtulowicz.[51] Man, aged thirty-two years. Syphilis ten years before. Increasing dyspnœa. Pain in suprasternal notch. Tracheotomy. No improvement. Sudden death. *Autopsy.* Annular cicatricial stricture at bifurcation of trachea admitting only a fine sound.

CASE XXII.—Beger.[52] (First case.) Man, aged thirty-three years. For three months cough, expectoration, dyspnœa, and cyanosis. No stridor. Pneumonia. Death. *Autopsy.* Some narrowing of trachea above bifurcation and of beginning of both bronchi. Large ulcer in trachea above stricture. Cylindrical dilatation of smaller bronchi. Scar on penis.

CASE XXIII.—Beger.[52] (Second case.) Man, aged thirty-five years. Syphilis many years before. Symptoms lasted five weeks. Cough, chills, fever, and increasing dyspnœa. Physical signs of intense bronchitis. Death. *Autopsy.* Small ulcer on posterior wall of upper part of trachea, with perforation of œsophagus. Inhalation pneumonia. Syphilitic lesions in testicles and elsewhere.

CASE XXIV.—Beger.[52] (Third case.) Woman, aged forty-two years. Syphilis for several years. For four months cough and increasing dyspnœa. Then stridor, cyanosis, suffocative attacks. Tracheotomy. Death. *Autopsy.* Old scars in throat. Both main bronchi greatly stenosed by enormous thickening of the walls. Dilatation of trachea above and of bronchi below.

CASE XXV.—Bernays.[53] Man. Syphilis ten years before. Some cough and dyspnœa. Sudden death from hemorrhage. *Autopsy.* Small ulcer of trachea perforating arch of the aorta.

CASE XXVI.—Oudin.[54] Man, aged thirty-five years. Syphilis five years before. Cough. Increasing dyspnœa. Intense stridor. Tracheotomy. Death in six months. *Autopsy.* Diffuse gummatous swelling of wall of lower end of trachea and of both bronchi, with marked stenosis. Many small ulcers, with submucous fistulous tracts.

CASE XXVII.—Schroetter.[31] Man, aged thirty-two years. Cough, with blood-stained expectoration. Ulcer seen in trachea, which healed under mercurial inunctions. Two months later scar had formed a membranous stricture of extreme grade. This was gradually relieved by catheterization through the larynx.

CASE XXVIII.—Ewart.[55] Woman, aged thirty-eight years. Evidences of old syphilis. Chronic bronchitis for many years. Died of "pulmonary and renal disease." *Autopsy.* Evidences of syphilis in

liver, spleen, and throat. Extensive scars throughout trachea and both bronchi. Stenosis of orifice of left bronchus. Interstitial pneumonia.

CASE XXIX.—Sturge.[56] Boy, aged two and one-half years. Inherited syphilis. Intense dyspnœa and stridor. Cyanosis. Whispering voice. Sudden death. *Autopsy.* Extensive ulceration and cicatrization of larynx. Beginning stenosis of upper part of trachea.

CASE XXX.—MacKenzie.[57] (First case.) Boy, aged ten years. Inherited syphilis. For several weeks cough, aphonia, and increasing dyspnœa, with suffocative attacks. Loud inspiratory stridor. Inspiratory thrill, greatest over lower part of trachea. Laryngoscope showed ulceration of larynx and redness and bulging of tracheal wall. Almost complete cure under mixed treatment.

CASE XXXI.—MacKenzie.[58] (Second case.) Man, aged forty-five years. *Autopsy.* Extensive old scars in middle portion of trachea. Pneumonia of right upper lobe. Old tuberculosis of one apex and of bronchial glands. Syphilitic scars over skin and in pharynx. Periosteal thickening of tibia. Cirrhosis of liver.

CASE XXXII.—MacKenzie.[59] (Third case.) Young man. Syphilis five years before. For eighteen months increasing dyspnœa, at first only inspiratory. Paralysis of both abductors of the larynx. Tracheotomy unavailing. Death. *Autopsy.* Ulcers and gummatous infiltration at lower end of trachea, with marked stenosis. Both recurrent laryngeal nerves involved in a mass of cicatricial tissue and enlarged bronchial glands. Large gumma at apex of lung.

CASE XXXIII.—Gleitsmann.[60] Woman, aged thirty-two years. Syphilis for several years. Gradually increasing dyspnœa, with a single suffocative attack. Aphonia. Laryngoscope showed two membranous bands in trachea, opposite fifth or sixth cartilage, forming a sort of false glottis and causing great stenosis. Cured by KI.

CASE XXXIV.—Jacobson.[61] Woman, aged forty-three years. Evidences of old syphilis in throat. For several weeks increasing dyspnœa, with paroxysmal attacks. Stridor. Tracheotomy. Temporary improvement. Death. *Autopsy.* Cicatricial stricture of trachea at fourth and fifth rings. Emphysema. Chronic congestion of organs.

CASE XXXV.—Lancereaux.[62] Man, aged forty-one years. Syphilis fifteen years before. Cough. Dyspnœa. Aphonia. Stridor. Blood-stained sputa. Emaciation. Death. *Autopsy.* Cicatricial stricture of lower end of trachea and of both bronchi. Stricture of larynx. Dilatation of bronchi. Syphilis of liver.

CASE XXXVI.—Semon.[63] (First case.) Woman, aged forty-six years. Evidences of old syphilis. Pain in throat. Cough. No dyspnœa. Voice normal. Laryngoscope showed large ulcer in upper part of trachea. Cured by KI.

CASE XXXVII.—Semon.[63] (Second case.) Girl, aged sixteen years. Severe syphilitic manifestations in throat three years before. Cough, increasing dyspnœa and stridor. Laryngoscope showed extensive reddish mass beginning at fourth ring and almost filling lumen. Complete cure in six weeks under mixed treatment.

CASE XXXVIII.—Kopp.[64] (First case.) Man, aged thirty-three years. Syphilis six years before. Cough, fever, chills, emaciation, increasing dyspnœa. Loud stridor. Suffocative attacks. Tumor

mass seen near lower end of trachea. Death. *Autopsy.* Enormous thickening of walls, and stenosis, of lower part of trachea, and of both bronchi. Dilatation of bronchi. Interstitial pneumonia.

CASE XXXIX.—Kopp.[64] (Second case). Woman, aged sixty-five years. For two years cough, hoarseness, increasing dyspnœa, pain in throat, stridor. Tracheotomy. Death. *Autopsy.* Ulceration of larynx and trachea. Stenosis of trachea and left bronchus by extensive fibrous endotracheitis and peritracheitis.

CASE XL.—Schech.[65] (First case.) Man, aged thirty-four years. Chancre fourteen months before. Severe cough, night-sweats, increasing dyspnœa, stridor, suffocative attacks. Sputum contained connective and elastic tissue. Gradual recovery under KI.

CASE XLI.—Schech.[65] (Second case.) Man, aged thirty-five years. Syphilis eleven years before. For one year cough, increasing dyspnœa, night-sweats, stridor. Laryngoscope showed at the lower end of trachea a nodular, dark red tumefaction, obstructing lumen of trachea and right bronchus. This mass disappeared under KI.

CASE XLII.—Davidson.[66] Man, aged thirty years. For five months cough, expectoration, tracheal pain, increasing dyspnœa, stridulous breathing. Death. *Autopsy.* Large, irregular ulcer at lower end of trachea. Right bronchus much constricted by scar tissue. Evidences of syphilis in liver, spleen, and testicle.

CASE XLIII.—Ingals.[67] (First case.) Man, aged forty-five years. Syphilis seven years before. Increasing dyspnœa, stridulous breathing, paroxysmal cough, *tirage*, suffocative attacks. Area of dulness to left of sternum. Larynx and upper half of trachea normal. Under large doses of KI marked improvement. Sudden death several months later. Cause unknown.

CASE XLIV.—Ingals.[67] (Second case.) Man. Syphilis six years before. For two months violent coughing attacks, severe dyspnœa, stridor, *tirage*. Stricture located at lower part of trachea by auscultation. Gradual improvement under the iodides.

CASE XLV.—Ingals.[67] (Third case.) Man. Hoarseness, cough, and dyspnœa on exertion. Laryngoscope showed several small ulcerating gummata in the trachea one-half inch below the larynx. Later, the formation of cicatricial bands causing stenosis. Tracheotomy. Under antisyphilitic treatment stricture gradually disappeared. In six months tracheotomy tube could be removed.

CASE XLVI.—Massei.[34] Woman, aged twenty-eight years. History of probable syphilis. For two years dyspnœa and cough. Inspiratory and expiratory stridor. Laryngoscope showed in the trachea at the third ring a diaphragm-like membrane, with an oval opening admitting a No. 12 French catheter. Much improvement by catheterization.

CASE XLVII.—Dreschfeld.[68] Man, aged thirty-nine years. Symptoms for eight months. Cough, increasing dyspnœa, stridor, *tirage*, cyanosis, orthopnœa. Death. *Autopsy.* Small ulcer in middle part of trachea. Fibrous thickening of tracheal wall as bifurcation, with stenosis of both bronchi. Bronchopneumonia. Syphilis of liver.

CASE XLVIII.—Gulliver.[69] Woman, aged thirty-one years. Syphilis seven years before. Died of "bronchitis and pleuropneumonia." *Autopsy.* Annular stricture of trachea above bifurcation. Scars of old ulcers in trachea and both bronchi.

CASE XLIX.—Schuman-Leclerc.[70] Woman, aged thirty-three years. Died with diagnosis of pulmonary and intestinal tuberculosis. *Autopsy.* Extensive ulceration and cicatrization in lower part of trachea. Ulcers in both bronchi. Larynx normal. Lobular pneumonia. Gummata of liver. Deforming endarteritis. Amyloid disease of spleen.

CASE L.—Turner.[71] Man, aged forty years. Destruction of soft palate several months before. Sudden fatal hæmoptysis. *Autopsy.* Large ulcer in trachea just above orifice of right bronchus, with perforation of superior vena cava. Ulceration also of both bronchi.

CASE LI.—Bésançon.[63] Man, aged sixty-two years. Syphilis forty years before. Symptoms chiefly those of emphysema. No stridor. No evidence of stricture. Death of pneumonia. *Autopsy.* Stricture of lower part of trachea. Much narrowing of bronchus to left upper lobe. Scars in liver.

CASE LII.—Gotthelf.[73] Woman. Doubtful history of syphilis. For four months cough, expectoration, and increasing dyspnœa. Loud stridor. Tracheotomy. Death. *Autopsy.* Two large ulcers in lower part of trachea. Below these an abscess in wall causing stenosis of trachea and left bronchus. Left bronchus and branches dilated beyond stricture.

CASE LIII.—Gougenheim.[74] Man, aged sixty years. Syphilis twenty years before. Increasing dyspnœa, with loud stridor. Voice clear. Larynx normal. Orthopnœa. Symptoms much improved by antisyphilitic treatment.

CASE LIV.—Parker.[75] Boy, aged fifteen years. Many signs of inherited syphilis. Increasing dyspnœa. Inspiratory stridor. Clubbing of fingers. Died eventually of acute catarrhal pneumonia. *Autopsy.* At one and a half inches above bifurcation begins an "obliterating endotracheitis," with enormous thickening of the tracheal wall *within* the cartilages. Same condition found in large bronchi. Left bronchus almost occluded.

CASE LV.—Silcock.[76] Man, aged thirty-two years. Syphilis eight years before. Symptoms for four months. Cough, expectoration, pain between the shoulders, emaciation. One hæmoptysis. Death from pneumonia. *Autopsy.* Tracheal mucous membrane reddened and infiltrated. Many small ulcers. Ulcers throughout larger bronchi. Bronchi generally somewhat dilated. Pneumonia of right lower lobe. Gummata of liver.

CASE LVI.—Fränkel.[77] (First case.) Woman, aged forty-one years. Hoarseness, cough, emaciation, weakness. Signs of consolidation at left apex. Paralysis of left vocal cord. Death from exhaustion. *Autopsy.* Many irregular gummata throughout the lower two-thirds of trachea. Scars throughout trachea and both bronchi. Bronchi, bronchial glands, and left recurrent nerve embedded in a mass of dense fibrous tissue—"diffuse indurative peribronchitis."

CASE LVII.—Fränkel.[77] (Second case.) Woman. Symptoms developed insidiously. Rupture of œsophagus in coughing attack. Death from inhalation pneumonia. *Autopsy.* Large area of ulceration in trachea which at one point has perforated the œsophagus. Interstitial hepatitis. Amyloid disease of spleen and kidneys.

CASE LVIII.—Lucas.[78] Woman, middle aged. Syphilis seven years before. For three months pain and tenderness in upper part of

trachea. Some cough and dyspnœa. Laryngoscope showed rounded, reddish mass on anterior wall of trachea just below larynx and almost filling lumen. Disappeared under KI.

CASE LIX.—Moure. (Mauriac.[79]) Woman, aged forty years. For several months increasing dyspnœa, loud inspiratory stridor and cough. Voice normal. Laryngoscope showed tracheal mucous membrane swollen, dark red, and covered with inflammatory exudate. Complete cure in one month by mixed treatment.

CASE LX.—Fauvel. (Mauriac.[79]) Patient only seen in an attack of suffocation requiring tracheotomy. Intense stridor. Death in a few hours. *Autopsy.* Large gumma situated astride the point of bifurcation of trachea and causing great obstruction.

CASE LXI.—Gulliver.[80] Woman, aged thirty-eight years. For three months cough, increasing dyspnœa with paroxysmal attacks, stridulous breathing. Death. *Autopsy.* At lower end of trachea and in both bronchi many irregular, deep ulcers and much scar tissue. Stenosis of trachea and left bronchus. Pneumonia of left lung. Extensive scars in soft palate.

CASE LXII.—Sokolowski.[81] (First case.) Boy, aged nineteen years. Cough and gradually increasing dyspnœa for three years. Later, suffocative attacks. Loud stridor. Voice clear. Larynx moves only very slightly on inspiration. Died in suffocative attack after tracheotomy. *Autopsy.* Diffuse hypertrophic and cicatricial thickening of tracheal wall with diminution of lumen. Inner surface granular, scarred, and ulcerated. Both recurrent nerves embedded in a mass of indurated bronchial glands.

CASE LXIII.—Sokolowski.[81] (Second case.) Man, aged fifty-six years. Syphilis twenty-five years before. Cough, gradually increasing dyspnœa, loud stridor. Gradual development of paralysis of both laryngeal abductors. Tracheotomy. Temporary improvement. Later suffocative attack. Death following a second hæmoptysis. No autopsy. Author believes case one of tracheo-bronchial syphilis, with probably enlarged glands and compression of the recurrent nerves.

CASE LXIV.—Gairdner.[82] Woman, aged thirty-eight years. For several months cough, paroxysmal dyspnœa, blood-stained sputa, inspiratory stridor. Death. *Autopsy.* Ulcer three and a half inches long in lower part of trachea, perforating wall at one point and extending into both bronchi. Inhalation pneumonia. Gummata of liver.

CASE LXV.—Landgraf.[83] Man, aged forty-six years. Syphilis for twenty years. Cough, increasing dyspnœa, suffocative attacks. Tracheotomy. Death. *Autopsy.* Extensive syphilitic changes in pharynx, epiglottis, and larynx. Ulcers and scars in lower part of trachea and in both bronchi. Purulent peribronchitis. In right bronchus was found a portion of cricoid cartilage which was lacking from larynx.

CASE LXVI.—Raymond.[84] (Lécureuil.[17]) Man, aged fifty-four years. Syphilis eighteen years before. For three months cough, emaciation, night-sweats, increasing dyspnœa. Intense inspiratory stridor. Tumor mass on right side of trachea. Death. *Autopsy.* Dense fibrous and caseous mass partly surrounding and compressing the upper part of trachea. Compression of right recurrent nerve. Syphilomatous nodules throughout trachea. Pneumonia. Gummata of liver.

CASE LXVII.—Lancereaux. (Lécureuil.[17]) Man, aged sixty-two years. Syphilis for forty-two years. Cough, expectoration, dyspnœa,

nocturnal suffocative attacks. Death. *Autopsy.* Scars and stricture at lower part of trachea. Recent stricture at level of cricoid cartilage. Stenosis of bronchus to left upper lobe. Bronchopneumonia.

CASE LXVIII.—Mader.[85] Woman, aged fifty-one years. Cough for two years. Increasing dyspnœa. Stridulous breathing. *Tirage.* Pain in suprasternal notch on swallowing. Death. *Autopsy.* Extensive ulceration in middle portion of trachea. Fibrous stricture of lower end of trachea and of right bronchus. Interstitial pneumonia. Dilatation of bronchi. Syphilis of liver.

CASE LXIX.—Davezac.[86] (Favraud.) Woman, aged seventy-three years. Evidence of old syphilis in nose and throat. Dyspnœa, stridor, *tirage*, suffocative attacks. Death. *Autopsy.* Fibrous strictures of both main bronchi.

CASE LXX.—Wright.[25] (First case.) Woman, aged thirty-three years. Cough, blood-stained sputa, emaciation, fever, increasing dyspnœa, suffocative attacks. Tracheotomy. Death two and a half months later. *Autopsy.* Whole of trachea irregularly thickened and ulcerated. Cartilages necrotic. Interstitial pneumonia of left lung, with dilatation of bronchi.

CASE LXXI.—Wright.[25] (Second case.) Woman, aged forty-five years. For three months cough, and blood-stained sputa. Later dyspnœa and suffocative attacks. Tracheotomy. Death. *Autopsy.* Trachea ulcerated throughout. Great stenosis two inches below larynx. Pneumonia.

CASE LXXII.—Parrain.[30] (First case.) Woman, aged forty-nine years. Cough, blood-stained sputa, increasing dyspnœa, loud stridor, nocturnal suffocative attacks. Laryngoscope showed larynx normal. In trachea a reddish swelling projecting into lumen from right wall. Cure under mixed treatment.

CASE LXXIII.—Parrain.[30] (Second case.) Man, aged thirty-nine years. For four months increasing dyspnœa, intense stridor, *tirage.* Breathing inaudible over left lung. Larynx normal. No relief from antisyphilitic treatment. Death. *Autopsy.* At lower end of trachea and in right bronchus the mucosa is congested and softened. Stenosis of left bronchus from enormous thickening of walls.

CASE LXXIV.—Griffin.[87] Woman, aged thirty years. Syphilis eight years before. For some months tickling in throat and cough. Increasing dyspnœa, with paroxysmal attacks. Laryngoscope showed in upper part of trachea a large, red, irregular, projecting mass covered with exudate and obstructing lumen. Cured by antisyphilitic treatment.

CASE LXXV.—Kayser.[88] Man, aged twenty-five years. Syphilitic ulceration in throat. Small tumor in neck below larynx. Irregular ulcer seen on anterior wall of trachea. On pressing tumor air and fluid can be seen issuing from tracheal ulcer. Under KI tracheal ulcer healed and tumor diminished in size.

CASE LXXVI.—Seifert.[35] Man, aged forty-three years. For one year gradually increasing dyspnœa; slow, labored breathing; stridor; *tirage.* Feeble respiratory signs over left chest. Emphysematous ballooning of apex of left lung. Laryngoscope showed larynx normal, slight stenosis of trachea at third cartilage, and marked stenosis just above bifurcation. Physical signs determined stenosis of left bronchus. Both tracheal and bronchial strictures gradually relieved by catheterization.

CASE LXXVII.—Nixon.[89] Man, aged forty years. Evidence of old syphilis. Increasing with symptoms of laryngeal stenosis. Larynx seen to be normal, however. Death. *Autopsy.* Great constriction of lower end of trachea and of both bronchi by gummatous mass surrounding trachea and bronchi.

CASE LXXVIII.—Aufrecht.[24] Patient showed signs of tracheal obstruction, including absence of the normal tracheal breath sounds heard by stethoscope. Gumma the size of a plum seen just above bifurcation. Under KI symptoms were improving when patient died of pneumonia. *Autopsy* showed gumma to be almost completely broken down.

CASE LXXIX. Duplant.[90] Man, aged sixty-one years. For one year gradually increasing dyspnœa, weakness and cough. Suffocative attacks upon any slight exertion. Tracheal stridor heard by stethoscope. Sudden death. *Autopsy.* Trachea normal. Extensive change in wall of left bronchus, with marked stenosis. Thickening of the perichondrium, ossification of cartilages, disappearance of muscle and elastic tissue, cicatrization of mucous membrane. Pleurogenous interstitial pneumonia of left lung.

CASE LXXX.—Hanzel.[91] Woman, aged forty-eight years. For five months cough, blood-stained sputa, and increasing dyspnœa. Stridor on exertion. Larynx normal. In trachea just below larynx is seen a rounded, red, tumor mass almost filling lumen. Rapid cure under KI.

CASE LXXXI.—Ovize.[92] Woman, aged thirty-six years. Syphilis nine years before. For six months cough, expectoration, emaciation, and increasing dyspnœa. Slow, labored breathing; stridor; *tirage;* suffocative attacks. Larynx normal. In trachea is seen a small gumma at level of third cartilage. Slow but marked improvement under mixed treatment.

CASE LXXXII.—Woman, aged thirty years; single and a servant. Admitted to the Hudson Street Hospital on December 3, 1901. Family history good. Aside from an attack of some gastric disturbance one year ago, which lasted for one month, patient says she has always been healthy. She gives no history of rheumatism or syphilis. Admits an excessive alcoholic habit.

Present illness began only two weeks before admission, when, after exposure, she had a slight chill and began to cough and to suffer from dyspnœa. The cough was accompanied by expectoration of white, tenacious mucus which was at no time blood-stained. Dyspnœa became so marked that the patient was obliged constantly to sit up in bed in order to breathe. There was some pain on coughing. These symptoms had steadily increased in severity.

On admission temperature was 104° F.; respiration, 36; pulse, 120.

Physical Examination. The patient is a woman of medium stature, muscular, and fairly well nourished. She sits upright in bed, with head well forward and anxious, pale face. Skin and mucous membranes are moderately cyanotic. No subcutaneous œdema. Voice is feeble and rather husky.

The respirations are moderately rapid (30 to 36). Inspiration is prolonged, labored, and accompanied by a slight but distinct stridulous sound and by slight sinking-in of the tissues in the supraclavicular and suprasternal regions and of the epigastrium. Expiration is shorter and not labored.

Lungs. Expansion feeble over both chests and apparently equal. Vocal fremitus is unsatisfactory because of the light, feeble voice. Percussion gives everywhere a somewhat hyperresonant note except in the lower part of the left axilla, where the resonance is diminished. Nowhere is an area of well-defined dulness to be found. All the lung borders extend considerably beyond their normal limits.

Auscultation shows the vesicular murmur everywhere feeble, but especially so over the lower lobes, where it is almost entirely masked by sibilant and sonorous rhonchi. A few fine moist râles are heard at both bases posteriorly. Vocal resonance faint and unsatisfactory.

Heart. Apex indistinctly felt in the fifth intercostal space 9 centimetres to the left of the mid-sternal line. The normal area of absolute cardiac dulness is entirely obscured by the voluminous lungs. Heart sounds are faint and clear. Action rapid, regular, and somewhat feeble.

Both liver and spleen are distinctly enlarged, and the free borders of each can be felt a finger's breadth below the costal border. In all other respects the abdomen seems normal.

The labored inspiration and its slight stridulous sound suggested some laryngeal obstruction, but a careful laryngoscopical examination showed no abnormalities in the larynx, save some slight hyperæmia of the cords, nor could any obstruction be seen in the trachea.

The larynx moved only slightly with respiration. The head was not retracted. No struma or enlarged glands could be detected in the neck. The urine contained a trace of albumin.

During the next three days the condition of the patient showed little change. The dyspnœa and cough rather increased in severity, while the temperature fell to about 101° F. and remained there.

The symptoms were so suggestive of stenosis of the larger air passages that the chest was repeatedly examined for evidences of aneurismal or other growth which might explain the symptoms.

On December 7th, four days after admission, there were two paroxysmal attacks of suffocation. In the second of these the pulse failed rapidly, the temperature became subnormal, and the patient died.

Autopsy. (Dr. Biggs. Condensed report.) *Chest.* Pleuræ show a few old adhesions on both sides. No fluid. Pericardium normal.

Heart of normal size. Cavities partly distended with fluid blood. Valves competent. Muscle of fairly normal color and consistence. Coronary arteries show a few small areas of arterio-sclerosis.

Lungs. Left. Lung tissue shows everywhere a condition of marked emphysema. The small bronchi contain mucopurulent secretion. A portion of the anterior half of the lower lobe is consolidated, of reddish color, and has the appearance of an area of lobar pneumonia. *Right.* This shows the same condition of emphysema. A considerable portion of the middle lobe is in the condition of gray hepatization. In the central part of the upper lobe is a small area of beginning consolidation.

Larynx normal.

Trachea and Bronchi. Slight uniform thickening of tracheal wall everywhere. More marked at lower end, where wall measures 4 mm. in thickness. At a point 8 mm. below bifurcation the *left* bronchial wall becomes greatly thickened, with such encroachment upon the lumen that this measures only 2.5 mm. across. This thickening of the

bronchial wall involves the entire circumference of the bronchus and extends downward for a distance of 1 cm., where the wall resumes its normal thickness and the lumen widens out into a cylindrical dilatation of 13 mm. diameter. On section through the thickened wall no cartilage can be seen, and the wall has a thickness of from 1 mm. to 8 mm., the tissue being, apparently, entirely dense fibrous tissue.

In the *right* bronchus just below its first division there is a similar stricture, 1 cm. long and from 3 mm. to 4 mm. in diameter, involving the branch to the lower and middle lobes. The wall here is 5 mm. to 6 mm. thick. This stricture on section shows the same absence of cartilaginous rings and accumulation of fibrous tissue as does that of the left bronchus.

The wall of the right bronchus shows some general thickening (3 mm. to 4 mm.) in addition to that producing the stricture. The bronchus is not dilated beyond the point of stenosis.

Spleen. This is about one and a half times its normal size and is very firm. On section it shows the typical "sago" bodies, and with Lugol's solution quite extensive amyloid degeneration is seen.

The *liver* and *kidneys* also show a moderate degree of amyloid degeneration, but in other respects show little departure from the normal.

In none of the other abdominal viscera were lesions of importance to be found.

Aorta. Throughout the whole of the aorta and the larger arteries generally the intima is thickly studded with nodular plaques of arteriosclerosis, some of which show atheromatous degeneration and calcification.

Microscopic Examination. (Conner.) Sections of the thickened and strictured portions of the two bronchi show practically identical lesions. The normal bronchial wall is entirely replaced by fibrous tissue, in which no trace remains of either muscle or elastic tissue. The mucous glands have disappeared, and of the cartilaginous rings nothing remains but a few small fragments of rarefied cartilage situated in the centre of the fibrous mass. The columnar epithelium is replaced by a number of layers of cuboidal and flattened cells which approach as they near the surface the type of pavement epithelium.

At the periphery of the wall are to be seen small lymph nodes infiltrated with fibrous tissue and closely adherent to the fibrous mass. The fibrous tissue is dense and old and shows scattered through it are many small areas of round-cell infiltration and a few thick-walled bloodvessels.

Elsewhere throughout the bronchi and trachea the moderate thickening of the walls seems to be due to an increase in the fibrous layer enclosing the cartilages and in the submucous coat.

The nature of the bronchial lesions themselves, the amyloid degeneration of the liver, spleen, and kidneys, and the extensive arteriosclerosis (at the age of thirty years), seemed to leave no doubt that the strictures were of syphilitic origin, although no definite syphilitic history had been obtained from the patient.

The character and distribution of the bronchial lesions may be indicated, perhaps, by the cumbersome name of *diffuse indurative peribronchitis and endobronchitis.* As to the manner of origin of the condition only surmises can be made. It apparently did *not* arise from

the cicatrization of an ulcer of the mucosa. Whether it resulted from the formation of gummata in the bronchial wall, or in the bronchial lymph glands (as Lécureuil[17] has suggested), or whether, from the beginning, it was a diffuse productive inflammation of the wall, without gummatous deposit, must remain uncertain.

During the four days in which the patient was under observation no satisfactory diagnosis could be reached. The inspiratory type of dyspnœa, the faint stridor, and the inspiratory sinking-in of the neck and epigastrium, suggested some obstruction in the larger air passages. This was clearly, however, neither in the larynx nor in the cervical portion of the trachea. The physical signs of the thorax, also, failed to give any clue to the cause of the dyspnœa. Neither in the lungs nor in the mediastinum could any definite lesions, other than those of emphysema and bronchitis, be demonstrated.

In spite of the absence of all signs of thoracic aneurism that condition seemed to be, perhaps, the one most likely to be the source of the difficulty.

The fact that two considerable areas of pneumonic consolidation should have been altogether overlooked in the frequent physical examinations, was the source of surprise and mortification at the necropsy, until the lesions of the bronchi were discovered; then the absence of bronchial breathing was at once explained by the presence of the strictures.

Although the dyspnœa was quite severe throughout, the patient's condition was not regarded as grave until the appearance, on the last day, of the two suffocative attacks. Then, however, the dyspnœa became intense, the cyanosis grew more pronounced, and the heart failed rapidly.

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THE PROMINENCE OF THE EYEBALL AND A METHOD FOR MEASURING IT.[1]

By Edward Jackson, M.D.,
of Denver, Col.

The need of a simple practical method for measuring the prominence of the eyeball has doubtless been felt by many observers. The difficulties of estimating accurately the degree of exophthalmos, or enophthalmos, from Graves' disease, tumors, inflammatory swelling, and injuries to the orbit, or high myopia or hyperopia, have usually prevented an exactness in the reports of such cases that would be quite desirable. The lack of exact measurements also causes uncertainty regarding the progress of any given case.

Certain measurements of the orbit have been carefully made in the laboratory, and in the pursuit of various lines of scientific investigation. An elaborate instrument for measuring the prominence of the eye, suitable for laboratory studies, has been devised by Birch-Hirschfeld;[2] but we have had no fairly accurate and readily applicable method of determining the prominence of the eyeball suited to general clinical use. To meet this want I have worked out the following plan, which has for over three years served me in a very satisfactory manner.

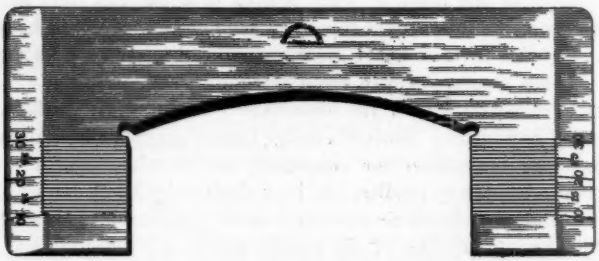
The point best suited for a reference point, in this connection, is the point in which a plane, passed through the visual axes of the eyes when these are in the primary position, intersects the outer margin of the orbit. This point is very nearly the most retreating portion of the outer margin of the orbit, and is easily determined with sufficient accuracy upon the normal face or upon the denuded bone. Hence it serves as a common point of reference for measurements made upon the living head and those upon the bare skull. Many measurements have shown that this part of the orbital margin has the least variable relation to the apex of the orbit, and to the more important landmarks of the skull. This point has a comparatively thin covering, which varies in thickness but little in different persons and in different con-

[1] Read before the College of Physicians of Philadelphia, June, 1902.
[2] Klinische Monatsblätter für Augenheilkunde, November, 1900.

ditions of embonpoint. The line joining these corresponding points of the two orbital margins constitutes a base line from which the prominence of the eyeball is to be measured.

To measure such prominence the rule shown in the illustration is employed. The curved side of the rule is pressed against the face, the straight edges resting on the outer margins of the orbits, so that the upper surface of the rule (the one shown in the figure) shall be just in the plane of the visual axes, the eyes being directed horizontally toward the distant object and the head held erect.

There are parallel lines on the upper surface of the rule. The observer places his eye to the right or left of the patient, according as he wishes to determine the prominence of the right or left eye. He then sights along the parallel lines and notices between which of these lines the most prominent part of the cornea appears to fall. The lines are ruled 1 mm. apart, and serve to indicate within 0.5 mm. the protrusion of the cornea beyond the line of reference—a degree of accuracy sufficient for all practical purposes.



Such an instrument, for measuring the forward projection of the eyeball, may be called a *protometer*. This name, suggested by my friend Dr. T. B. Schneideman, has an obvious relation to proptosis, which has come to be used chiefly in connection with the eye.

In making such measurements care should be taken to have the eyes turned pretty directly forward. A little deviation to the right or left will make but little difference in the result obtained; but a decided departure from the median direction will cause the eye to appear less prominent, because the centre of rotation is posterior to the centre of the anteroposterior axis of the eyeball, and because the diameter of the eyeball passing through the sclerocorneal junction is shorter than the anteroposterior diameter.

To provide for different widths of the face I have used two of these scales in question, one 96 mm. and the other 86 mm. in width between the two parts of the straight edge to be pressed against the orbital margin. For very young children a still narrower one might

be used; but for the great majority of adults a gap of 90 mm. in the straight edge is sufficient and answers the purpose. A rule of this kind can be quickly made from a piece of thin wood or cardboard. It has been neatly constructed of wood by Bonschur & Holmes, of Philadelphia.

In the great mass of cases the distance of the summit of the cornea in front of the reference line is between 12 and 20 mm., averaging about 16.5 mm. Often there is a difference of 0.5 mm. in the prominence of the two normal eyes. But rarely the difference amounts to 1 mm. unless due to disease, as excessive myopia of the more prominent eye, or orbital changes causing exophthalmos or enophthalmos. The averages indicate that the eye becomes a little more prominent up to the age of sixteen or eighteen years, and that there is a very little sinking in old age; but these changes, and those produced by wasting disease, are probably very much less than might be supposed from casual inspection of the appearance of the orbit. My intention, however, is to discuss these variations of both normal and abnormal eyes at some future time. The present purpose is to put before the profession this simple aid to exact diagnosis.

LOCALIZATION OF FOREIGN BODIES IN THE EYE AND THEIR REMOVAL:

WITH REPORT OF CASES.

By Louis C. Deane, M.D.,
SURGEON TO THE CALIFORNIA EYE AND EAR HOSPITAL, SAN FRANCISCO, CAL.

Localization of foreign bodies in and about the orbit has received a great impetus since the advent of X-ray diagnosis.

Formerly, if by any chance a foreign body, such as a piece of steel, iron, lead, shot, glass, porcelain, or coal, had penetrated into the paraocular tissues we were satisfied to know whether it was intra-ocular or extra-ocular, and whether, if it were a bullet, it had penetrated into the cerebral cavity. Occasionally we were able to locate it with the ophthalmoscope before the media had lost their transparency through hemorrhage, traumatic cataract, or inflammatory exudation.

If it could not be seen, one of three things had to be done: first, to thrust a magnet blindly into the vitreous, if there were reasons for believing it to be iron or steel; second, to remove the eye, if the inflammatory reaction were profound, or, if not, third, to allow the eye to remain, with the possible danger of sympathetic ophthalmia or in the hope that the foreign body might not be in the globe at all.

The sideroscope (a sensitive magnet hung on a strand of silk, which is deflected when brought near a piece of steel or iron) has a limited value in showing the presence of such a foreign body in the globe or orbital cavity.

The giant magnet of Haab, when brought within the distance of an inch of a piece of steel or iron, lodged in the eye, exerts a marked attractive force often sufficient to draw such a body to the front of the globe if it is not held by inflammatory adhesions or embedded in tissue, where it can be seen and extracted.

These methods of procedure, while they have their value, are often vague and uncertain, and I am convinced that frequently much unnecessary damage is done by the magnet where the exact location of an object is not known. Of course, these instruments are useless in the presence of lead, porcelain, glass, coal, etc.

An X-ray photograph of a foreign body lodged in the orbital region, when taken without the aid of a localizer, is most confusing if an effort is made to determine its position. It is for this reason that numerous attempts have been made to devise exact methods and instruments of precision for the determination of their exact location.

One method might be mentioned, that of Webster Fox, who places an oval band of gold, divided so as to correspond to the principal quadrants of the eye beneath the lid, its shadow acting as the localizer. Two skiagraphs are necessary, one a temporal and the other an occipito-frontal view.

I have tried this method, but must say that in my hands it was of little value. The same cases were subsequently localized by the method described in this article, and the decided and exact character of the results left no doubt in my mind as to its superiority.

The localizer here shown was constructed after the pattern of a machine used by Makenzie Davidson, of London, with the exception of some modifications to enlarge and render more accurate its field of usefulness. Its object is to determine the exact position of foreign bodies in the head with special reference to those lodged in the orbit and eyeball.

The great importance at times of calculating the exact position of a piece of steel, shot, or bullet, which has penetrated the orbital or cerebral cavity, and the difficulty, or I might say impossibility, of determining its exact location without the aid of a localizer similar to the one I present, prompts me to demonstrate the method whereby these results are arrived at, also to review a few characteristic cases with photographs and projections.

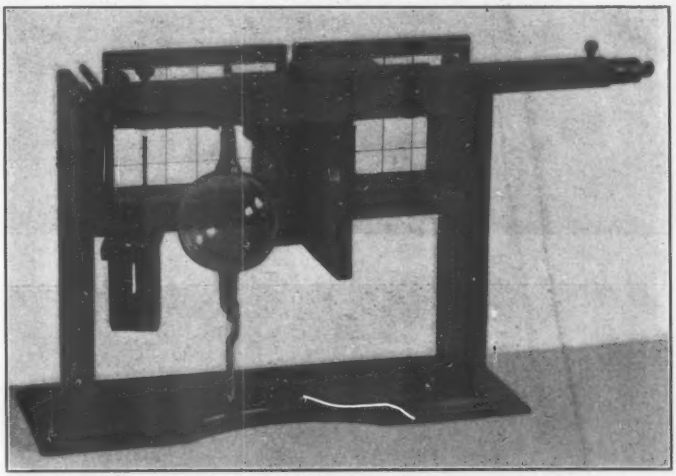
The method of taking two separate radiographs of an object held immovable, from two different points of view and calculating the

relative displacement of each in regard to certain fixed markings, to determine its distance from the plate, is not a new one.

This localizer is so constructed that it gives us exact figures to work upon, so that it is possible by the simplest methods of triangulation to arrive at exact conclusions.

The localizer consists of a framework supporting two cross wires at right angles to each other, and has a mechanism to hold a Crooke's tube in a certain position so that the luminous point of rays emanating from the anode is perpendicular to both. The head is then clasped in the machine and held immovable after proper localizers are placed on

FIG. 1.



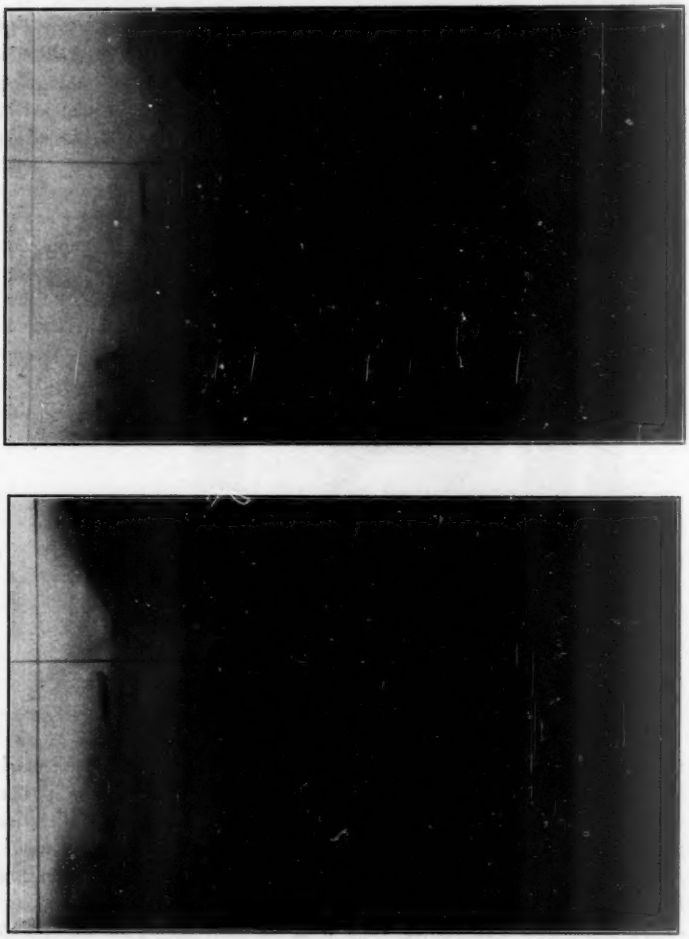
The localizer.

the face to determine the median line of the head, the vertical meridian of the eye, and the lower limbus of the cornea. The tube is then withdrawn on a line parallel with the horizontal wire a known distance and a radiograph taken. Without removing the head the tube is replaced in its original position and again withdrawn the same distance in the opposite direction, and another radiograph is taken.

We have now two radiographs with the same object shown on different parts of the plates, the distance between them and their relation to the cross-wires can now be easily determined. The distance of the luminous point on the anode from the plate and the displacement each way from the perpendicular are also known. The distance of the opaque markers, placed upon the face, from the plate has already been

measured to verify our results later. With this data at hand one may arrive at conclusions so accurate that the location of the foreign body

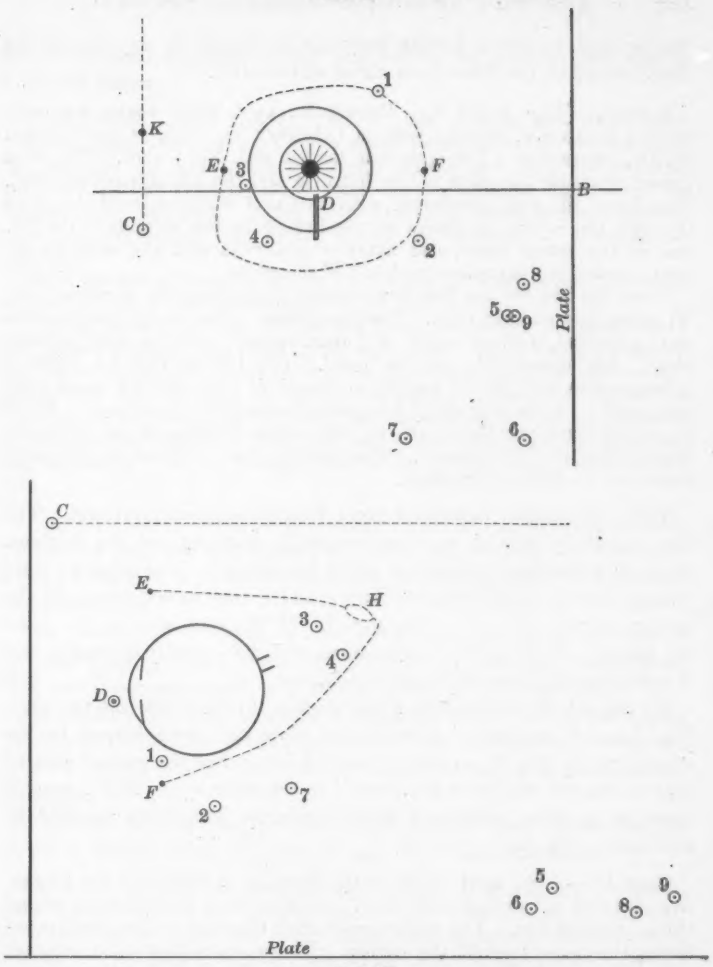
FIG. 2.



Case I. X-ray photographs (reduced in size) showing nine shot. The positions of the same shot in their relation to the cross-wires are shown in the two photographs. Their exact location in two planes is seen in Fig. 3.

can be determined within one millimetre. This localizer has a wider range of usefulness than the older instruments in that two-sized plates

FIG. 2.



Left Eye

Case I. Vertical and horizontal projections slightly reduced in size from the normal, showing the exact location of the nine shot. *C.* Lower end of nasal bones, which also marks the median line, corresponding to the nasal septum, vomer, perpendicular plate of the ethmoid, centre of optic commissure, etc. *D.* Wire placed on lower lid one millimetre to outer side of vertical meridian of the globe, the upper end of which corresponds with the lower limbus of the cornea. Shot 1, resting on the bone at upper and outer margin of the orbit. Shot 2, on the outer surface of the malar bone. Shot 3, near the apex of the orbit, resting below and to the inner side of the optic nerve; 13 mm. behind the globe. Shot 4, near the apex of the orbit lying below the optic nerve and 16 mm. posterior to the globe. Shots 5, 6, 8, and 9 are superficial, lying beneath the skin in the region of the ear. 5, 8, and 9 are about on a level with the auditory meatus, 5 in front and 8 and 9 immediately behind. No. 6 is near the tip of the mastoid, embedded in the attachment of the sternocleidomastoid muscle. Shot 7 is embedded in the masseter muscle over the ramus of the jaw. The actual distance of the various shots from the wire may be determined by a little descriptive geometry—1.36 mm., 2.28½ mm., 3.62½ mm., 4.57 mm., 5.13 mm., 6 9 mm., 7.32 mm., 8.8 mm., 9.11 mm. *D*, tip of the wire, 49 mm. *C*, point at lower end of nasal bones, 82 mm. *E* and *F* were determined by actual measurement upon the face; *E* was placed 18 mm. posterior and 13 mm. to the outer side of *C* (the lower ends of the nasal bones). The width of the orbit on a level with the horizontal meridian of the globe is 37 mm., and so *F* was fixed. The distance from *E* to the apex of the orbit was taken as 42 mm

can be used, so that a foreign body can be located in any part of the head instead of the immediate region of the orbit.

CASE I.—(Figs. 2 and 3.) Robert B., aged eight years, was shot from a distance of seventy feet on February 30, 1901. Case referred by Dr. Overacker. The gun was loaded with No. 7 shot. The shot passed through the skin of the left arm and the left side of the head. The lower lid was penetrated with two shot which entered the globe through the sclera at places corresponding to the wounds in the lid, one at the lower, inner, and anterior quadrant, and the other at the lower, outer, and anterior quadrant of the globe.

I saw the boy for the first time eleven days after the accident. L. V. perception of light only. Tension minus. The ocular conjunctiva was congested without signs of inflammation. Cornea and aqueous clear. Iris discolored, due to blood. The iris showed no signs of inflammation and dilated readily to atropine, none having been used previously. Lens and anterior capsule normal in appearance. With the ophthalmoscope there was no red reflex because of an extensive hemorrhage in the vitreous. The patient had suffered no pain, and there was no pain on pressure.

This case seemed to present some features of special interest. The shot evidently carried no infection with them, as not the slightest trace of inflammatory reaction could be noticed. The signs of their passage into the globe were conclusive, and if they passed out again the removal of the eye was not warranted. If the shot were in the globe the dangers of sympathetic ophthalmia would be greatly increased, and it would be well to consider its enucleation.

By the aid of the localizer I was enabled to show that the two shot, Nos. 3 and 4, had passed through the globe and were lodged in the orbital cavity (Fig. 3), so the eye was left intact and the patient assured that no danger was to be anticipated to the other eye. Had I been in doubt as to their location I would certainly have been justified in enucleating the eye.

CASE II.—J. S., aged thirty years, Spanish, referred by Dr. Payne, was shot full in the face with No. 7 bird-shot from a distance of about three hundred feet. The right eye showed that one of the shot penetrated the upper half of the cornea. There was a traumatic cataract with a part of the lens mass in the anterior chamber, and the iris prolapsed and incarcerated in the wound. There was a good deal of conjunctival œdema and a marked orbital cellulitis, with considerable pain. He could see moving objects on the temporal side.

The left eye showed slight orbital cellulitis. Anterior segment of the eye and lens uninjured, but impossible to get any view of the fundus. Absolutely no light perception. Eye tender, but no pain.

Both eyes gradually became softer and passed on to phthisis bulbi.

This case presents no special features, because both eyes were destroyed, but it demonstrated the location of a shot in the right eye, exactly midway between the horizontal and vertical meridians in the

lower and inner quadrant, 2 mm. anterior to the equator and resting upon the retina.

CASE III.—(Figs. 4 and 5.) Mr. L., two months ago, while hammering a pipe above his head, felt a sharp shock to his left eye, followed by pain for an hour. He bandaged his eye and continued working with no further inconvenience than sensitiveness to light when he removed the bandage. After four days the bandage was removed. The eye was blind, due to the rapid development of a traumatic cataract.

The case was referred to me by Dr. Payne for localization of a piece of steel or iron lodged somewhere in the globe.

The appearance of the eye at this time showed that a foreign body had passed through the cornea, iris, and lens. The anterior chamber had been evacuated, as there was an attachment between the corneal wound and iris, forming an anterior synechia. This had pulled the anterior layers of the iris forward, leaving the pigment layer adhesive to the anterior capsule of the lens, which was very misleading, giving the impression that it formed part of the foreign body lodged in the iris.

This case had already been placed before the giant magnet, and, while some pain was felt at the time, there was no real evidence that a piece of steel or iron was lodged there, and that method of extraction was abandoned.

The position of the piece of iron was determined by the localizer to be resting on the floor of the eyeball on a line with the vertical meridian and three and a half millimetres posterior to the equator.

An incision through the sclera was made in the immediate region of the point indicated in the diagram, and the foreign body extracted by the magnet, with a small piece of choroid in which it was evidently embedded.

I will say for the localizer that without its use I am certain that by no other method could this foreign body have been extracted.

CASE IV.—R. McL., aged thirteen years. Referred by Dr. Mays. Five weeks ago the boy was shot in the face with a load of bird-shot at a distance of forty-five feet. Many shot embedded themselves in his face. One passed through the upper lid and penetrated the eye through the upper ciliary region, evacuating the anterior chamber and prolapsing the iris, which was excised later. A severe uveitis was the result, the vitreous being entirely opaque. The lens, queer to say, is transparent, no traumatic cataract having formed.

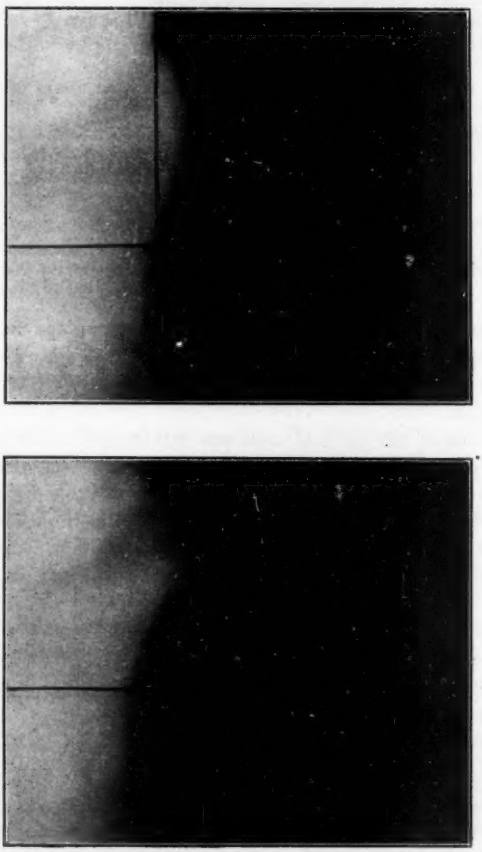
The eye is now sensitive, with pericorneal injection, posterior synechia. The patient is suffering from an orbital cellulitis with fever, which has confined him to his bed.

After localization, certain facts were elucidated. The shot that struck the upper lid is apparently not the one that passed through the globe. There are two shot in the orbit. The one that entered the globe passed out again and is lodged six millimetres posterior to it and rests upon the optic nerve. The one that passed through the upper

lid is in the upper part of the orbital cavity on a line vertical to the posterior pole.

Nine shot in all were located. The two shot in the orbital cavity had, no doubt, carried in the infection and were causing the cellulitis.

FIG. 4.



Case III. Shows the presence of a chip of iron lodged in the eye. Note Fig. 5.

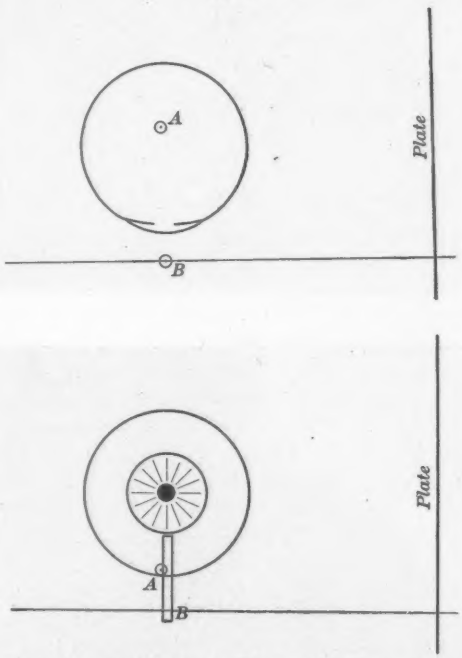
CASE V.—(Figs. 6 and 7.) P. D., aged forty years, miner. Referred by Dr. Pischel. Four years ago a piece of steel flew in the left eye, but later the patient picked up a piece of steel about as large as a pea on the floor. He could not see for two weeks, then good sight returned. A year later sight became dim again.

Two years ago a cataract was removed, and severe inflammatory re-

action followed. After the operation, until three weeks ago, he could see outlines of figures.

At present there is no perception of light. Cornea appears dim from fine, brown exudate on posterior surface. In the upper quadrant of the cornea half a millimetre from the limbus is a horizontal scar, to which the iris is drawn, so that the pupil appears vertically oval. Anterior chamber deep, iris trembling, and tension high. Fundus cannot be seen clearly enough to distinguish an excavation of the disk. Eserine lowers tension.

FIG. 5.



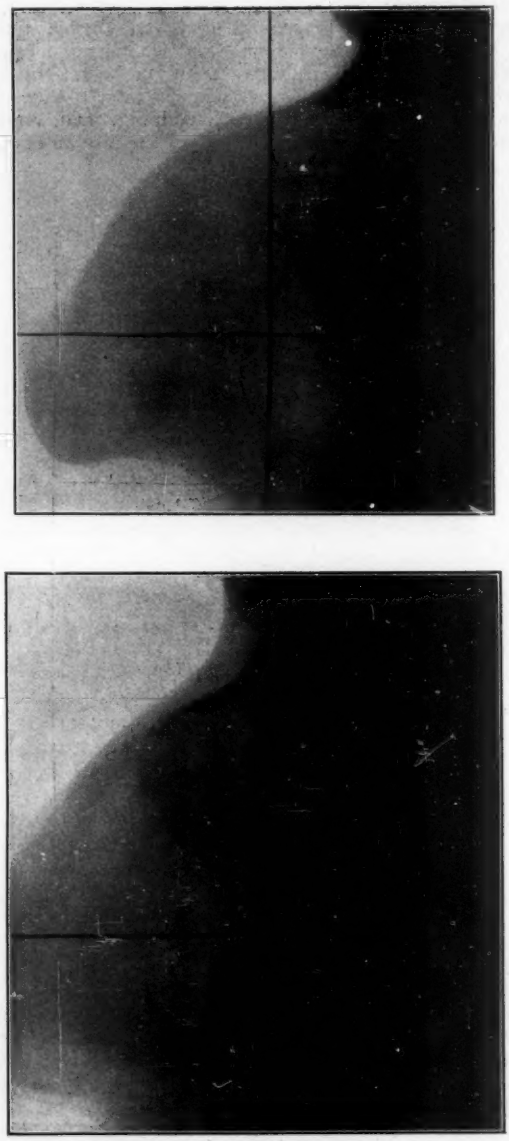
Case III. Horizontal and vertical projections showing location of chip of iron, *A*, as described in text.

The giant magnet caused considerable pain, indicating the presence of iron or steel in the eye, though it could not be dislodged.

After localization the foreign body was seen to be two and a half millimetres above the horizontal meridian, and five millimetres to the inner side and posterior to the limbus, resting upon the retina in the region of the ora serrata. An incision was made directly over this spot and the chip of steel removed by the magnet.

CASE VI.—(Fig. 8.) Referred by Dr. Brady. Seven months ago, while shoeing a horse, the patient's hammer missed the nail and struck the shoe, so that a splinter from the hammer struck him in the left eye.

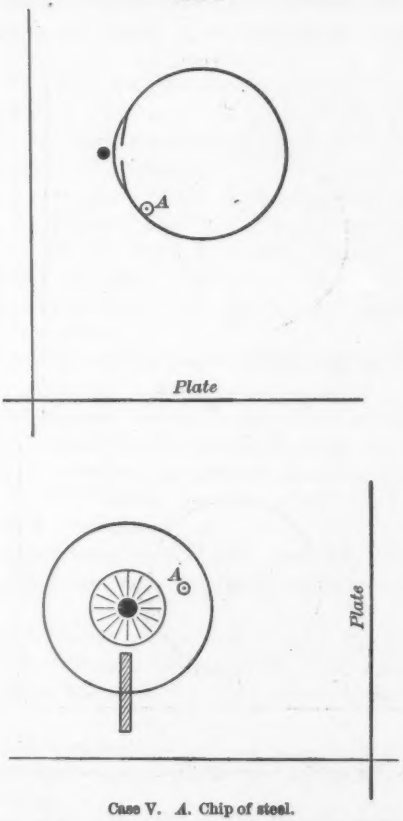
FIG. 6.



Case V. Chip of steel lodged in the eye. Note Fig. 7.

Dr. Brady saw him four hours later, when there were no inflammatory symptoms. At the middle of the inferior quadrant of the cornea, two millimetres from the limbus, was a linear perforation, and immediately behind it was a similar wound in the iris, about two millimetres long. Through the latter was seen the red fundus reflex and the margin of the uninjured lens. The fundus was normal, and the ophthalmoscope and oblique illumination failed to reveal a foreign body. The eye was several times approached to the giant magnet,

FIG. 7.



Case V. *A*. Chip of steel.

with negative results. As there was no reliable X-ray apparatus available at the time, Dr. Brady adopted palliative and antiphlogistic measures. Vision was 20/30. A few days later there was a mild iridocyclitis. The iritic congestion was depleted by instilling two drops of adrenalin chloride, 1 : 10,000, every two hours. Vision was now 20/200.

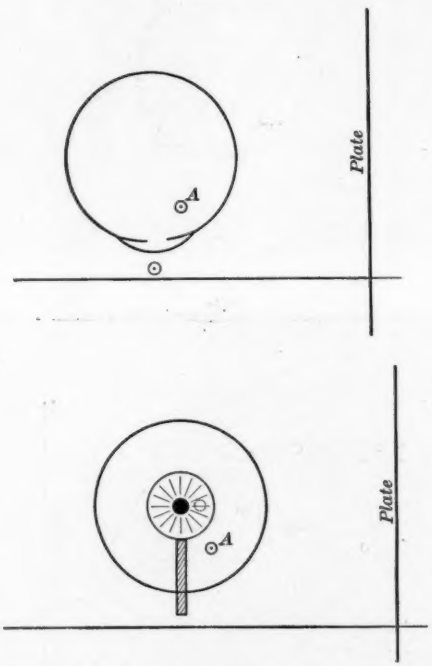
Having seen eyes enucleated and the foreign body found after failure with the magnet, Dr. Brady kept the case under observation.

For his own satisfaction and the patient's security he referred the case for localization, which showed a small foreign body, less than a millimetre in diameter, just behind the ciliary region and about the middle of the lower and inner quadrant.

The vision is now 20/50 plus, and the body being well tolerated, it was thought best to leave it undisturbed.

CASE VII.—(Figs. 9 and 10.) J. H., aged forty-nine years, blacksmith. Injured on May 10, 1902, and referred by Dr. Schloss. Was hammering one sledge upon another when something struck his right eye. Five days afterward there was marked chemosis, no iritis, lens

FIG. 8.



Case VI. Horizontal and vertical planes. *A*. Location of chip of steel.

opaque, no increase of tension. At first there was pain in the globe, which subsided before atropine had been used.

A piece of steel four by two millimetres was located two millimetres below the horizontal meridian and nine millimetres posterior and to the outer side of the limbus, resting lightly on the retina.

An incision through the sclera was made immediately over the part indicated in the diagram, and a large piece of steel extracted by the magnet, which verified both our calculations as to its size and position.

Résumé of Cases Showing Some of the Advantages of the Localizer. In Case I. nine shot were localized, but none in the globe, which showed two scleral wounds and a dense hemorrhage of the vitreous. The two shot that entered the globe passed out again. This dispelled the doubt as to their position and avoided possibly a subsequent enucleation.

Case II. shows how a great number of shot can be located on the same radiograph, and proves its accuracy, as the eye was removed and the shot in the globe was shown to be exactly as indicated in the diagram.

Case III. is of interest in that the giant magnet had been used without giving any positive evidence of the presence of a piece of steel, possibly because it was firmly embedded in the choroid and surrounded by organized inflammatory exudate.

An incision was made directly over the foreign body, with no other indication than the diagram, and by the aid of the giant magnet a piece of steel was removed through the sclera with a small mass of choroid and organized exudate.

Case IV. shows the location of nine shot and the undoubted cause of a severe orbital cellulitis.

Case V. A scleral wound was made directly over the foreign body, as shown in the diagram, and a piece of steel extracted.

The giant magnet was used without success, which shows its inability to dislodge a piece of metal when embedded in tissue, as in Case III.

Case VI. is only included among these cases because it demonstrates that very small pieces of metal, less than a millimetre in diameter, show themselves in the radiograph.

Case VII. is where a large piece of steel, four millimetres long by two millimetres broad, was removed by the magnet through a scleral wound.

The giant magnet was not previously used upon this case because it was deemed safer to extract it directly through the sclera than drag it through the vitreous, back into the ciliary region, possibly entangling it in the iris.

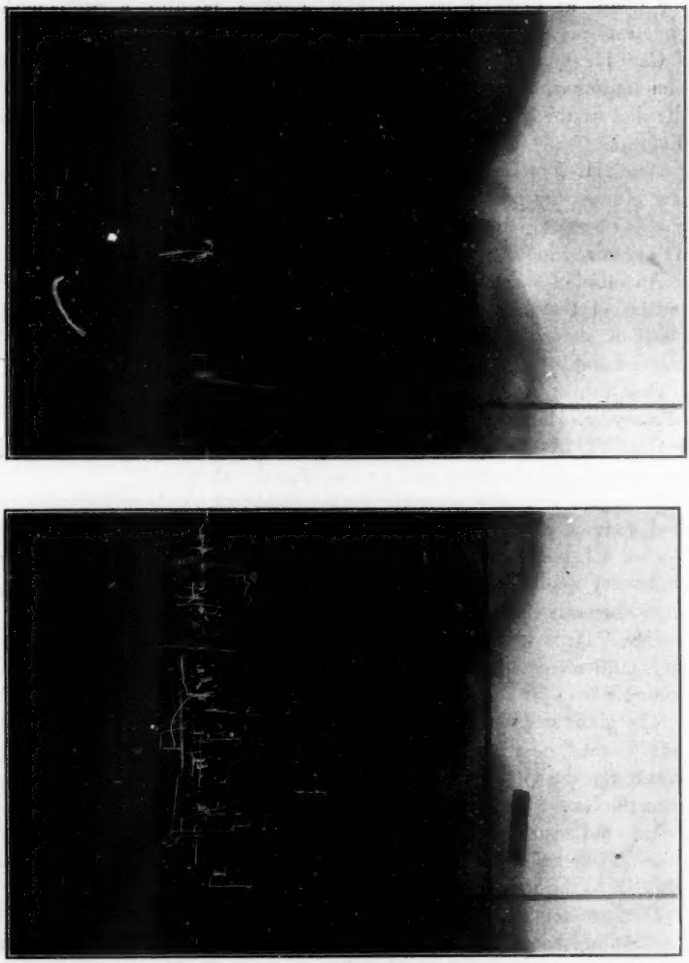
I do not wish to disparage the use of the giant magnet, for in many cases it has made such work possible, proving itself a most useful adjunct to the localizer.

It must certainly be apparent that if there is any doubt as to the location of the piece of metal, which must be the rule, the localizer should first be resorted to, thereby gaining valuable information, in many cases avoiding a considerable amount of unnecessary traumatism, and greatly simplifying its extraction.

The number of cases where the foreign substance can be seen are very few. Haab reports that in 165 cases 80 had traumatic cataract,

and, with hemorrhage and inflammatory exudation, it places this class of cases greatly in the minimum.

FIG. 9.



Case VII. The photographs show a piece of steel 4 mm. by 2 mm. in the orbital region. For its location, note Fig. 10 and the text.

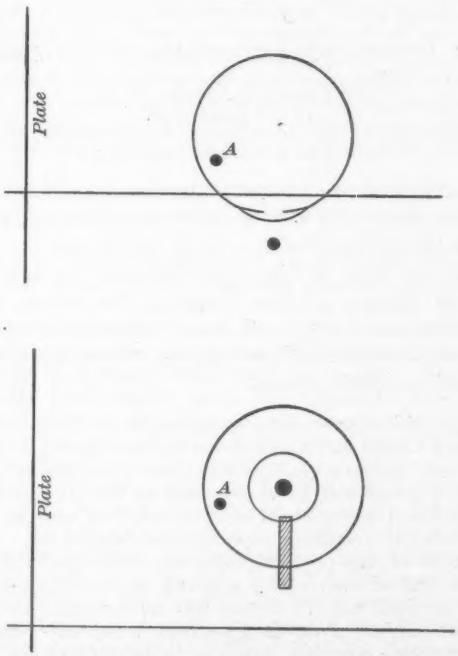
To show how easily one can be deceived by appearances, in Case III. a bit of iris pigment could not be distinguished from a piece of

iron by the closest observation. The localizer proved it to be pigment and showed a piece of metal in another place from where it was subsequently removed through a scleral wound, made directly over its location.

Some objection has been offered to removing foreign bodies through scleral wounds on account of the danger of detachment of the retina.

As to my own experience, in the past two years, with scleral wounds, I am pleased with the simplicity of this method, and in 14 cases thus treated I have not observed a subsequent detachment.

FIG. 10.



Case VII. *A*. Piece of steel. For a description of its location, note the text.

I have seen one case, previous to the present method of extraction, where a detachment of the retina followed the usual extraction of a piece of steel by the Haab magnet. The magnet was not used for three weeks after the injury, and, I believe, in drawing the steel forward it detached the retina to which it was adherent.

I am pleased to see that there are some who concur with me (Sweet, Holt, etc.) in the advisability of scleral extractions where the position

of the foreign substance is known and near or posterior to the ora serrata.

It must be said of many advocates of the giant magnet that they begin their work of extraction without exact knowledge of the position or size of the substance they are about to remove. To these the scleral wound is only a return to the old method of opening the sclera and inserting the tip of a small magnet blindly into the vitreous. With the localizer and giant magnet the latter need never be done, and there can be no question that much damage is thus avoided.

THE LYMPHATIC SYSTEM AND THE TONSILS.[1]

By Henry L. Swain, M.D.,
of New Haven, Conn.,
Professor of Diseases of the Ear and Throat, Yale University; Fellow of the American Laryngological Association, etc.

Permit me in presenting to your attention this evening this subject, with its rather uninviting title, to adopt the methods of our brethren who dispense the consolations of religion, and to take the history of a case as a text or point of departure, suggesting the lines of thought which will be followed in these remarks. The case is in itself rare enough to excite our interest, and demonstrates the reverse picture of what we have clinically long observed, viz., the influence of the tonsils on the general lymphatic system.

Mr. P., aged sixty-three years, consulted me in June, 1899, with the statement that he had had a bad throat for the last six months, having felt that he was having a gradual filling up of the throat without pain or soreness. He had had no violent cold or fever. His throat sometimes felt so full that he could scarcely swallow, and he often experienced difficulty in breathing, especially on lying down.

Examination of the physical condition developed that he was a fairly well-nourished man of his age, and apparently possessed a fair amount of physical vigor. There was no evidence in the general physical examination of anything, except what was referable to his throat, other than a complete alopecia, for which he wore a wig. The outside of his neck appeared to be somewhat thicker than seemed reasonable in a man of his general build, and the lymphatic nodes in the neck back of the angle of the jaw were diffusely but not greatly enlarged. Otherwise the glands of the body were palpable, but not increased in size. In the erect position he was able to breathe fairly well through his nose, or as easily through his nose as he did with his mouth open. Examination of the throat developed the condition which is evident in the drawing which I will pass around. Both faucial

[1] Read before the Section on Otology and Laryngology of the College of Physicians of Philadelphia, March 18, 1903.

tonsils were enormously enlarged, the increase in size being not merely toward the median line where they met, but in every direction, being circumferentially very much increased. The lingual tonsil, as is seen in the picture, was as much enlarged as I have ever observed, and seemed to not only project upward so as to be readily seen by direct examination, but also downward and backward sufficiently to depress the epiglottis, making it impossible to see the interior of the larynx. All this tonsillar tissue presented an absolutely pale surface. There was not the slightest trace of inflammation or other abnormality in appearance except the size and the pallor. Evidently we had to do with a perfectly simple hypertrophy. There could not be in the physical appearance a shadow of mistrust of anything in the way of tumor growth. Careful inquiry failed to develop that the patient had the slightest pain in any way whatsoever from this enormous mass, and he merely was annoyed because of the sense of fulness and the difficulty in swallowing and occasionally in breathing.

I immediately felt that we had here to do with a constitutional disease which was expressing itself in the tonsils, and to that end had an examination made of the blood, and for treatment started the patient upon the simplest of gargles and physiological doses of Fowler's solution.

In five days the tonsils had gone down considerably both in appearance and as far as the speech and comfort of the patient went. In five days more he could swallow with but little difficulty, and felt very much relieved. The examination of his blood had developed a moderate increase in the white blood cells present, a normal number of red blood cells, and a slightly diminished amount of hæmoglobin. Among the white blood cells there was proportionately a larger number of the small mononuclear type than one commonly sees. The eosinophilous cells were rather diminished in number. The polymorphonuclear cells were slightly if at all increased. The patient then disappeared; did not take his treatment, because the Fowler solution had begun to interfere with his comfort and digestion, and I saw no more of him for two months, when he came back with the condition of his throat even worse than on his first examination, and this in spite of having for two weeks taken Fowler's solution on his own initiative. He had, in fact, taken it in such large doses as to have again upset his digestion, and had failed to receive the relief which he had found so marked on his first visits. I then saw him at frequent intervals, at which I would spray him out thoroughly with extract of the suprarenal glands, and gave him some to use at home as a gargle. I had another blood count, and found that the blood conditions were similar to those of the previous examination. In a week of moderate dosage with arsenic and local treatment he had begun to improve, and by the end of a month he had gotten considerable space visible between the tonsils and uvula. At the end of two months he had gained fifteen pounds, and at the end of three twenty-eight pounds. All the tonsils had diminished very markedly in size, so that one could put two fingers if not three between the faucial tonsils. The lingual tonsil had sunk out of sight, so that it was hardly visible on direct examination; the epiglottis was raised so that one could see into the larynx, and the palate had come down nearly to its normal position, so that I was able to see the pharynx tonsil, which I had known to be increased

in size, by seeing it through the nose, and which was at one time nearly if not quite as large as was the lingual tonsil.

During these weeks that had elapsed by careful questioning of his family they had recalled some facts concerning his past history. As a young man of twenty or twenty-five years of age he had had lymph nodes swell to the size of a large hen's egg underneath one arm, and they had subsided again without external rupture. This happened within a few years certainly five or six times, and on each occasion with the same history of swelling, tenderness, threatening to rupture, but finally disappearing without any external break in the skin.

In October, when he first came back in such a bad physical condition, it had been quite easy to discover that his spleen was slightly enlarged, but no lymph nodes other than in the neck anywhere in the body seemed to be at all increased in size. An examination of the blood at the time of this great improvement showed it so nearly normal that it would have been very difficult to distinguish it from that of a perfectly healthy individual.

He was, however, improvident and careless now, as he had been all his life, ceased to take his medicine, and I saw nothing of him from February 23, 1900, until May 24th, when he returned with everything just as bad if not worse than at any time since the start. The tonsils absolutely met in the centre of the throat (this is the entry of July 3d), and for the last three months he has taken no arsenic whatsoever. He drifted along without a great deal of care, not presenting himself again until January 20, 1901. He again had taken no arsenic for three months. The tonsils were larger than it would seem possible could exist in the mouth of any individual. He now has a lump on the side of the neck under each sternocleidomastoid, and one at the end of the jaw, and on the right side one further down near the clavicle, and one under the arm. He was again started on the same regimé as before, when everything again seemed to improve; but on February 15th he reported as not having done so well lately in spite of regular treatment. The tonsils had rather increased, if anything, over what they were a month ago. In the substance of the hard palate there had come a swelling, hanging down into the mouth with spots of pressure necrosis. The lymph nodes all over the body were enlarged. He became very weak, and from this time on was confined to the house.

During the previous November he had a pneumonia, which ran a course of one week and left him prostrated in bed for the two succeeding. While in bed he noticed a swelling on the left side of the head in front of the ear, which increased in two weeks to the size of a walnut. Shortly after this a swelling appeared over the mastoid process of the right temporal bone. These and the swollen condition described in the palate remained until he died. The blood was now found to contain a great many more white blood cells than formerly, but nothing like the condition of enormous increase which one sometimes sees in lymphatic leukæmia.

Through all of this his kidneys, bowels, and other internal organs seemed to operate perfectly successfully and comfortably. By February 14th in front of the left ear a tumor the size of a hen's egg rather rapidly developed, freely movable all the time under the skin. The tumor at the back of the ear increased in size. The difficulty in swallowing now proved to be a serious impediment, and adequate nutrition

could not be accomplished. It was suggested to give rectal alimentation, but the patient bore it very poorly. Fowler's solution, while accepted and borne as far as the stomach was concerned, seemed to have absolutely no effect, and the patient gradually grew weaker, until he died from exhaustion, on March 4th, about two years from the time when he first noticed his tonsils enlarging.

Other than the slight pressure necrosis which occurred in the tumor in the hard palate nothing approaching breaking down or the formation of pus took place in any of the nodes. The family refused an autopsy, but we were allowed to excise a portion of one of the nodes of the neck and of the tumor in the mouth, and they revealed by microscopic study simple lymphoid hypertrophies. In the specimens as we were able to get them there was no regularity of structure, the lymph-node formation having apparently been so interfered with in the rapid growth as to be obliterated. A very careful study of the cells, I am sorry to say, was not carried out, but in general I am confident that there was no increase in the large polymorphonuclear cells or in any except the small lymphocytes. The tissue was not stained for tubercle bacilli.

Plainly here, it seems to me, we have had to deal with a general systemic trouble expressing itself first and foremost in the tonsils, the entire ring of lymphoid tissue being involved. This is quite different from the case to be briefly mentioned in a moment of acute Hodgkin's disease, beginning by infection of the tonsils. It is, in fact, the absolute reverse. In an individual formerly subject to adenopathies came a gradual and persistent enlargement of the tonsils. The appearance, as has been stated, was entirely different from the usual hypertrophies, and there surely was nothing of the inflammatory type. Then the marked diminution in size under Fowler's solution was so evident that nothing more was needed to demonstrate that the enlargement was due to constitutional dyscrasia. Also the fact that during his periods of improvement the blood reached normal, and his increase in weight and other changes showed how certainly the general system was responsible for the condition of the tonsillar tissues. Hence, we are justified in concluding that we here had to do with a case of lymphadenoma,[1] the first tissue to be affected being the tonsils, and it seems to demonstrate what oftentimes has been overlooked and forgotten, that these collections of lymphoid tissue known as tonsils are not merely what the word "lymphoid" derivatively signifies—*i. e.*, like or resembling lymph tissue—but really are integral parts of the lymphatic system.

This case, then, comes with peculiar force to demonstrate this connection, and would seem, therefore, to present unanswerable arguments if the question of the function of the tonsils was under consideration.

[1] There was nothing about the patient suggestive of tuberculosis, and yet it is exactly in this kind of a patient that the latter is present sooner or later.

It will be remembered that radical observers have arisen who contend that the tonsil is always a pathological product. If so, then, are all lymph nodes, Peyer's patches, and solitary follicles in the intestine abnormal? We have long had evidence that systemic conditions influence the tonsil, such as the diminution in size of the whole lymphoid tissues and marked falling off of the number of cells in the follicles, and also great lessening of the emigrants to the surface in profound wasting diseases, as, for example, chronic suppuration.

The case referred to earlier proved how overwhelming an infection from tonsil tissue can be when a young man of twenty is suddenly stricken with acute Hodgkin's disease following a simple streptococcus tonsillitis and dies within six days. Evidently there must have been some direct opening into the lymphatic current, and this we know exists just as surely in the tonsils as in any other lymph node. In this particular case the result could not have been more rapid had a pure culture of germs been injected directly into the lymphatic node or tonsil.

These two cases, each in its own way, demonstrate to my mind the absolute "oneness," to borrow a term from the disciples of Mrs. Eddy, of the general lymphatic system and the tonsils, even if we lacked other and more objective anatomical proofs. An inflammation of the tonsils in the one case fatally diseases the whole lymphatic system. In the other case a fatal disease purely of the lymphatic system swells the tonsils as the first nodes to become diseased, and then the others all over the body.

To speak of objective or anatomical proofs, it is known to all that structurally the tonsils are like the lymph nodes in almost every detail, only that they have no afferent lymph vessels. They are terminal nodes with lymph vessels leading away from them to the main lymphatic trunks, and so their cell elements go directly into the circulation as do those of any other lymph node.

If one were to attempt to sum up the physiology of the tonsil it might be succinctly stated that the functions of the tonsils were those of the lymph node, only that these particular nodes absorbed directly from the mouth and pharynx instead of taking the fluids brought to them from the surrounding tissues.

Analogous conditions exist in other parts of the body, such as, for example, the well-known process of absorbing the contents of the intestines directly into the lacteals. Here fluids are absorbed directly into the lymphatic trunks, and they go immediately to the general lymphatic current, explaining toxæmias arising from the intestine along similar lines with those produced by the tonsils.

Just why the tonsillar ring exists where it does is not quite clearly evident. It apparently represents a meeting-place of the embryonic

epiblast and hypoblast, and is early in evidence in the young life of the embryo, fully equipped for work when the child is born. Early testimony of the absorptive power occurs in the enlargement of the lymph nodes lying on the anterior surface of the spinal column, and suppuration of these makes the well-known cases of retropharyngeal abscess. Probably the absorption is from the pharynx tonsil. Later, the anatomists tell us, these nodes disappear, and usually in the adult there is only one large lymph node, which is on the ventral surface of the atlas, and the absorbed materials go to the other nodes, than in the young babe, probably to those in the region of the hyoid bone. With design and purpose even in the earliest days of the life of the individual the tonsils begin their work of absorption, and so may be reasonably concluded to normally exist with the intention of the Creator rather than as a purely pathological product.

All this and more could easily be believed to be but following the natural lines of thought in the application of the simple theme suggested by our text. Very well-worn truths many of them, I hear you say, but may they not for all that bear once more the light of your intelligent scrutiny? It has seemed to me that we have all along missed something somewhere in explaining the different conditions as we meet them in childhood. Simply because an event is an every-day occurrence is no reason why we should not try to explain it. Quite the contrary. What constitutes the condition known as "delicate," always catching cold, never strong, present, and most in evidence, perhaps, in the childhood of an individual? If he lives to adult life he never is free from it, and when hoary with age he folds himself in abundant wraps to supply what his system should have inherently been able to do in the way of protection from draughts and colds. Have we not come, general practitioners and specialists alike, to think of the tonsils as a concrete entity, an excrescence in the throat? If they get big and in the way, chop them off, any way to get rid of them, and then, proud in the consciousness of a righteous act bravely done, do we not forget that often but a small projecting fragment has been removed? Do we not as serenely contemplate our work as thoroughly accomplished as if we have pulled a tooth or ablated a wart! Or, put the picture the other way. We glance into the throat at these excrescences, the tonsils. They are seemingly not in the way, and we dismiss them as thoroughly from our minds as of no interest to us and certainly of no moment to their host, and as easily as we might a wen or a mole. But anon the victim in question is sick; temperature, fever, violent headache. All that he has is a little spot on one tonsil—a very trifling thing, but fraught with what consequences? It may be a diphtheria, septic poisoning results; the patient dies of heart failure. It may be a streptococcus inflammation and produce general joint rheumatism, endo-

carditis, or, as already referred to, acute Hodgkin's disease. It may be in a young child, and the tonsil inflamed be the pharynx tonsil up behind the palate, out of sight, where we may not even see the spot, and the result is not only a mighty disturbance of the general system, but the cervical lymph nodes enlarge, perhaps later the supraclavicular, and later still the bronchial. Perhaps one or another of these enlarged nodes stays and holds up a tubercular germ. For the time being it may completely encapsulate and swallow it up, and yet years after, when something overwhelms the host in the pride of young womanhood or manhood, the germ is let loose into the system and there results a fatal, quickly progressing tuberculosis.

Why is it that such an overwhelming majority—over 70 per cent.—of all children in foundling institutions, when dead from any cause, have enlarged bronchial nodes? What is it that happens when the young child, after the simplest of these acute seizures in the third tonsil, goes around for months, perhaps years, pale, seemingly anæmic, yet not demonstrably so, easily knocked out by the slightest affection, the nodes in the neck often swollen and tender even after the original seizure is long passed, never seeming to get quite up to the measure of resistance which was formerly his or hers? Or, let us reduce the matter to its simplest terms. Why should a great, healthy, robust adult be so completely felled by a few small follicular spots on this unimportant excrescence, the tonsil? If we ever stop to ask ourselves the question I fear we have all too often fallen into the habit of the old country philosopher, when some new problem was presented to him, of giving the lucid explanation, "It's a way things has." Tonsils certainly do have this way of doing, and in most instances that explanation will suffice. But when we are considering as to-night, can we not go a step farther on along logical lines and say that it is easy to thus poison the system, because when the tonsil is diseased a part of the lymphatic system—*i. e.*, the blood-making or blood-cleansing system—is involved; hence, therefore, the blood itself? A hypodermic injection gives maximum effects from minimum doses, because it is absorbed directly into the circulation. When any germ or poison enters the tonsil, unless arrested there or in the near-lying nodes, it eventually enters the blood stream, getting maximum effects from a very small area of disease.

Is there any anatomical reason why the results of tonsillar infection are more widespread as regards the lymphatic system in children, as a rule, than it is in adults? Of course, we are bound to bear in mind, when asking this question, the general statement that immature tissue resists less vigorously than that better organized and older.

When that distinguished observer Sappey, whose monumental work on the lymphatics will last for all time, could not get good injections of the lymph vessels in any given part, he learned to go to the newborn

babe or very young infant. Here he could get results with greater ease, because the channels were open, the vessels were fully developed, and many of the beautiful plates illustrating his atlas were thus made possible.

Therefore, it is easy to see how much greater freedom to run riot within the lymphatic system the poisonous or diseased substances have in the infant over the adult, and why the lymph nodes themselves have a great burden thrown upon them to filter off material thus easily gaining an access to the lymph current, and, if you will, how very dependent the future of that individual is upon the activity and intactness of these nodes and their physiological functions!

Now, of all the parts of the lymphoid ring the pharynx tonsil is the one most frequently attacked in the young infant, and, therefore, inflammations of it are most liable to endanger the general system. There is some evidence that the pharynx tonsil is the earliest to acquire full development. In any case it is the one which at birth stands ready for business. So it is reasonable to suppose that it would be the one to most frequently become diseased in the early years of life—a fact long known clinically.

We have led up logically to this point as perhaps of chiefest importance, certainly, to my mind, most vital; and if I am allowed to say anything further I should like very briefly to dwell on the question of acute inflammation of the pharynx tonsil in children.

Only within the last two years has one been able to observe any attention as paid in current literature to this acute inflammation of the pharynx tonsil. So much has been written about the chronic enlargement that the acute seizures have been passed over, largely because it is only lately that we have begun to recognize some of their far-reaching consequences.

Time does not permit me to give you any extensive description of this acute inflammation. That I have already done in another place; but I would like to emphasize the fact that some of the cases are easily missed and yet most significant in their results.

It is superfluous to speak before this audience further of what is well known to all of you of the typical, well-developed acute cases of tonsillar inflammation in the nasopharynx, but there are certain of these masked cases that escaped, and may for some time to come fail of intelligent consideration. The briefest way to mention the matter to-night will be to relate an absolutely typical masked case, and make a few critical remarks upon it in closing what is even now a lengthy paper.

I was called last April to attend a child whom I had often seen before. He possessed to my knowledge slight tonsillar enlargement, but was not by any means a mouth-breather, nor did I know of any-

thing wrong in his nasopharynx. He had, at the time of my visit, a high fever, headache, and slight snuffles; no cough, no sore-throat; slight ringing in the ears. Patient impressed one as being sick and demanding intelligent attention, and so was looked over very thoroughly. I examined the chest, tonsils, nose, and larynx, and could find as the only spot which showed trouble a very red, very moderately swollen, slightly spotted third tonsil. I gave my opinion that the cause of the trouble was there and there only.

I saw the patient the next day, and his fever had gone up in the afternoon higher than on the previous day, with a morning remission to nearly normal. There was no change in the physical condition except that the head ached harder. There was no cough, and the only sign of discharge from the nose on this second day was a slight sticky condition of the mucus around the entrance to the nostrils. The family physician was called in order that he might discover something that I might have omitted, and accepted with considerable mental reservation my diagnosis. He proposed, as I was very thankful that he did, to give the patient a thorough overhauling again. He found absolutely nothing that he could conscientiously say was productive of the symptoms, and on the following day, when the fever still persisted, not any higher, but with less remission, he, fearing that there might be something more in the case than we had either of us supposed, had the blood examined, made another thorough examination of the whole body, and still nothing developed.

During the following three days the temperature stayed considerably above 100°, but no other identical symptoms developed except that the lymph nodes in the neck at the angle of the jaw and further down under the sternocleidomastoid muscle became slightly puffed, very soft, and quite tender. Some weeks of general malaise and slight febrile rise at night followed. He dragged around with no ambition, and could not be made to pick up in weight, and the family made an early trip away to Maine for the summer. It took him some months to establish anything like his former vigorous condition.

If this were a single instance it would seem hardly probable that we had ruled out every possible condition leading to general dyscrasia, but it has happened again and again in other patients where it was well known that the adenoids existed and where these acute attacks, slightly different from the present, had been frequently enough observed to know where to lay the blame.

In this case the patient was old enough and tractable enough to allow of a posterior rhinoscopy, and there was no doubt of the existence of this follicular inflammation in the pharynx tonsil.

Through some three or four years that I have been in my own mind developing some of the ideas here expressed, I have had occasion to be intimately associated and to know of numerous such cases, and it has left in my mind not a doubt as to the fact that we had to deal later in this case with a distinct interference with or disturbance of the function of the lymphatic system, and it has developed therefrom that we might possibly explain why certain children would for years and years possess

the same characteristics as these patients surely have shown during the months or years that some of them have been under observation. And if this is so, if any of the deductions that we have made from this are even in part true, then it has become high time that the profession as a whole, and we, all of us, should view the tonsil as not merely an irresponsible, functionless lump of matter existing in the throats of our patients, but as an organ which, if not doing its full physiological duty or if diseased, can modify and influence some of the most vital functions of the lymph and blood. So, therefore, we who deal with the subject from the specialist's point of view can least of all afford to place ourselves in a position which shall seem inappreciative of these long-known and well-recognized physiological principles; for by so doing we throw ourselves open to the oft-repeated taunt that we take too narrow a view of our work, failing in many instances to at all consider the general system as we ablate tonsil after tonsil.

What should be the practical deduction from such theorizing as we have indulged in during these remarks? Surely, it is not to give free rein to indiscriminate attempts at removal of tonsils first, second, or third, and surely it would not argue that we must let them alone. It seems to me the practical point is that when we operate, to be thorough in what we do, and when it is done, be it ever so thoroughly, it must still be borne in mind that we have some tonsillar tissue left. As tonsillar tissue is normal to the human body, therefore, the patient thus operated upon has all the chances that a normal tissue always has of becoming infected or diseased. We have, therefore, not given our patients immunity because we have operated; we have only given them less liability, and we should, in the line of what we are now saying, never dismiss patients as needing no further thought, because, forsooth, they have received our skilful manipulations in the nasopharynx or pharynx. Surely, there is a broad field for progressive work along the lines of strengthening the bulwarks which nature has endeavored to erect that there may not happen to the little people in our charge some of these dire disasters, which have been mentioned in the early part of the paper, as coming from trifling affections of the tonsils. How can these be avoided? At present the way does not always seem quite clear, but it will never be if we do not occasionally ponder over the matter and endeavor to clear as much of the mist of doubt as is permitted to us, each in his own way and time.

THE INDICATIONS FOR OPERATIVE INTERVENTION IN MIDDLE-EAR SUPPURATION.

By Barton H. Potts, M.D.,

ASSOCIATE IN DISEASES OF THE EAR IN THE PHILADELPHIA POLYCLINIC; AURIST AND LARYNGOLOGIST TO ST. MARY'S HOSPITAL; AURIST AND LARYNGOLOGIST TO THE OUT-PATIENT DEPARTMENT OF THE GERMAN HOSPITAL; ASSISTANT OPHTHALMIC AND AURAL SURGEON TO THE CHILDREN'S HOSPITAL, PHILADELPHIA.

When to operate or to advise operation in cases of chronic suppurative otitis media is a question that is constantly being asked of the aurist, and it is one that is not always easily answered. Many patients live to a ripe old age without any serious trouble from a discharging ear; but it is without doubt that many have died from an overlooked or neglected aural condition whose lives might have been saved by operative interference. A discharging ear is not now treated with the same indifference as formerly; it has become more widely recognized that the ear is a part of the body, and that disease of this organ may affect the general health as much as disease of any other organ.

Intracranial complication is, of course, the condition to be dreaded, and, while it is comparatively infrequent, it is always dangerous. It is impossible to make up a complete list of the cases of otitis media that have been complicated by intracranial involvement, but the apparent proportion is increasing as the advance in aural work is being better understood and the diagnosis more scientifically made.

The chief point in our diagnosis is to determine the cause of the discharge. If the latter consists chiefly of stringy, ropy mucus, we will almost surely find the exciting cause to lie in the Eustachian tube or nasopharynx; but if the discharge is seropurulent or a creamy pus, and especially if it is very profuse and of long duration, we must look to the tympanum or mastoid; and it is this type of case that is referred to in this article. No definite rule can be made for these cases, for each must be studied upon its own merits. The discharge in itself is not much of a guide to us, except that a very profuse and long-continued flow would point to the involvement of a greater area than that comprised in the tympanum alone. Careful search must be made for necrosed bone within the tympanum, and if found this should be treated on proper surgical principles. Curetting must be done and thorough drainage established; if these measures as done through the canal fail, more radical treatment will be necessary, and the question of an operation on the antrum or mastoid must be decided.

When we have a large fluctuating swelling behind the auricle our course is generally plain; but when there is no swelling or induration, when even tenderness over the mastoid may be absent, our course of

procedure may not be so clearly evident. Too much importance should not be given to the symptom—for it is only a symptom—of fluctuation behind the auricle. Having eliminated the possibility of glandular involvement or of a furuncle or other inflammation of the external ear, we know that this swelling is a symptom of inflammation in the antrum or mastoid cells; but that this inflammation may exist in the deeper tissues without the presence of swelling has been proven very frequently. Many cases have been seen in which this swelling disappeared coincidently with a free flow of pus from the canal; unfortunately, this is regarded by the patient, and sometimes by the physician, as a great improvement and they rest satisfied until further, and generally more serious, trouble follows. Its true meaning is simply this: the pus has found a passageway for itself through the tympanum or through a sinus and thus evacuated the abscess cavity; but the inflammation of the deep tissues, which is the cause of the pus, still continues active. To consider a suppurative mastoiditis well, or even improved, simply because the swelling has disappeared is a decided mistake.

Local tenderness is an important sign in these cases; careful palpation will, in the majority of cases, elicit tenderness over the inflamed region, the point of greatest tenderness usually being found over the antrum, and persistent tenderness in this locality is a pretty sure sign of inflammation of the deep tissues. Tenderness over the mastoid tip is not of such great importance, except in those cases in which the pus has forced its way through the inner wall of the tip into the digastric fossa.

But, though usually present, tenderness may be entirely absent; this is more apt to be the case when suppuration is of very long standing. In these cases we are almost sure to find that a sclerotic process in the mastoid has changed completely the character of the bone; instead of cellular structure we may find a dense layer almost as hard as ivory; this change may be confined to the outer table alone or may extend to the antrum, and this may be obliterated, though not usually. This layer of dense bone may so effectually protect the inflamed deeper parts that we may be unable to elicit any tenderness even by the firmest pressure. By making pressure first over one mastoid and then over the other, the patient can sometimes by contrast aid in our diagnosis. Œdema behind the auricle will probably be found to be due quite often to furuncle or other inflammation of the external ear, and it will be difficult to determine the cause of the swelling. A point mentioned by Randall will aid us, *i. e.*, if in making our pressure over the mastoid and carefully avoiding the auricle we get pain, we may feel certain that the trouble lies in the mastoid; but if the pain is felt only when moving the auricle, we will find the trouble somewhere in the external ear.

One of the most important symptoms of all is an inflamed, tender, and bulging upper posterior canal wall and membrane. This condition in chronic suppurative otitis media is an almost certain indication of antrum or mastoid inflammation, and the failure of this condition to improve under treatment is one of our surest indications for operation.

The state of the general health is a guide in some cases, especially in adults. Children seem to be able to carry pus in the mastoid with much less apparent effect on their health; they are frequently seen with a history indicating a mastoid involvement extending over a considerable period of time, yet showing little or no impairment of health. But, unless there be very free drainage, an adult will generally show constitutional symptoms within a short time, and the sudden and rapid failure of health, with a history of chronic suppurative otitis media, is a very significant sign.

The temperature in adults is not so important as in children; the former will have pus in the mastoid for a considerable time and present little or no elevation of temperature, while a child will rarely have pus for any length of time without showing some pyrexia. This would not apply to pyæmic cases or those having a septic thrombus, for in these cases the extreme variations of temperature is the most significant sign of all, whether the patient be a child or an adult; here we have the temperature that is characteristic of pus, fluctuating rapidly through a range of four, six, or even eight degrees. When the sinus is thrombosed we frequently find tenderness extending back toward the occiput, and there will often be some bogginess just below the occipital bone. Tenderness along the jugular must be carefully searched for, as it is an important diagnostic sign in these cases.

Intracranial complications are the ones to be most dreaded; their development is generally so gradual and often so free from symptoms, that it may not be until an advanced stage has been reached that they come under the aurist's care and a diagnosis is possible. Even under careful observation the condition may not be certainly determined, and it is not until the inequality or the sluggishness of the pupils, rapid failure of strength, rigors or convulsions, elevation or subnormal reduction of temperature and slowness of pulse occur that the diagnosis is made with any degree of certainty. The variations of temperature and slowing of the pulse is the most characteristic sign and the one most often noted, and when present usually indicates intracerebral pressure. Von Bergmann (*Arch. f. klin. Chirurgie*, vol. xxvi.) says the position of brain abscesses of otitic origin is typical, viz., either in the temporal lobe or in the hemisphere of the cerebellum of the diseased side.

In regard to the clinical importance of the micro-organisms concerned in these processes the various authors differ. Randall teaches

that the majority of the acute infections are due to the pneumococcus, and that this organism is the most prone to cause meningitis and other intracranial affections of a toxic type; while the bone lesions generally show the streptococcus or staphylococcus either as the primary or complicating infection, which is the worst in mixed cases. Gorham Bacon (*Manual of Otology*) has long insisted on the importance of the streptococcus, and has been foremost in America in urging routine bacteriological examinations.

The presence of a sinus leading into the mastoid, whether from behind the auricle or along the canal wall, is a positive indication for operation. Masses of granulation tissue along the canal wall should always be studied with great care, for they are suggestive of exposed bone or a sinus.

Facial palsy is not of much significance, for we know that it occurs in many cases in which there is no mastoid involvement; but that this symptom may be caused by a suppurative mastoiditis was demonstrated to the writer, who had the pleasure of seeing a ten-day-old palsy clear up within seventy-two hours after operation.

Epileptiform attacks are very unusual, but they were present in one of the writer's cases, so that it was for relief from this symptom that the patient applied for treatment and not on account of the discharging ear.

Even with the above points borne carefully in mind, cases will be met in which it will be difficult to determine the question of operation. Some authors go so far as to say that it is a mistake to try to cure chronic suppurative otitis media by the milder methods of antiseptic douching, curetting of the tympanum, etc.; but in the opinion of others this extremely radical position does not find favor. Certainly many cases have been seen in which the discharge ceased under the local treatment and the ears have remained dry during a number of years. Under present methods the operation is so nearly free from danger and from disastrous after-effects, that except from a cosmetic standpoint some aurists prefer to operate and find it to have been unnecessary rather than to fail to operate in one case where it may have been needed.

The treatment in the cases of mastoiditis complicating chronic suppurative otitis media must be vigorous from the start. If the mastoid involvement is acute the first and most important step is to put the patient to bed and keep him there at perfect rest. He should be put upon a light diet and given a saline purge. Calomel is very favorably regarded by some men, especially if the mastoiditis is due to an acute otitis media. Its full administration, however, must be restricted to those patients of a fairly robust habit.

The application of leeches over the mastoid has been pretty generally

abandoned, as it has been found much more efficacious to secure local depletion by the very free incision of the membrane and canal wall, so as to secure profuse bleeding and relieve tension in the antrum. This opening into the tympanum should be made very early, not only to secure depletion, but also to permit of free drainage. To secure relief from this condition without resort to more radical measures, it is absolutely necessary to establish free drainage. Following this incision hot douching is one of our most efficient remedies; to accomplish results this douching must be copious, frequent, and hot. It is usually well to begin with a temperature of 110° F. and increase this to 112° to 114° F., using not less than half a pint at each time. As little force as possible should be used, the water being allowed to gently flow in and out of the ear, and it is usually well to douche at first every hour for several times and then every second or third hour. In the interim of hot syringing the application of dry heat over the mastoid is of value; this is best applied by means of a hot-water bag, hot salt or hop bag; but moist applications, such as poultices, hot onions, etc., are to be strictly avoided. Protection by compress and bandage should be continuous between douches; external heat being applied outside of it.

The application of cold over the mastoid is sometimes beneficial, but should be used carefully and, as a rule, its use should not be prolonged beyond forty-eight hours. The application should be continuous, and if, after removal, there should be a return of the symptoms it would not be advisable to reapply it, for it would mean that the cold is simply relieving the symptoms, but that the inflammation is still present, and that more radical measures are necessary. Some authors say that the cold should never be used in cases of streptococcus infection.

Various measures are recommended by different men, but opinions as to their usefulness are contradictory; and the mercurial and saline, the early paracentesis and hot douching seem to give much the best result. When the milder methods of treatment fail more radical ones must be adopted; but nobody wishes to have his mastoid opened unless it is absolutely necessary, so that this should be held as a later resort. On the other hand, too long delay is attended with much greater danger than too early intervention, for the opening of the mastoid cells through to the antrum is not in itself a dangerous procedure, and the opinion is very generally held that in obscure cases, when treatment has failed, the exploratory opening into the antrum is indicated.

What shall be the form of operation is an important question to decide. The consensus of opinion seems to be that the original Schwartze operation—opening through the mastoid cells into the antrum—is sufficient only in those cases when the mastoiditis is acute and without the history of chronic suppurative otitis media; but when

the latter is present it is necessary to more thoroughly clean up the attic and antrum and give more free and lasting drainage. When the mastoid cells are not involved, this is best accomplished by the Stacke operation, which consists in laying forward the auricle, as in the regular mastoid operation, and separating the membranous from the bony canal by a blunt instrument; a portion of the upper posterior canal wall is removed and all parts of the tympanum, viz., the antrum, attic, and tympanum proper, are thrown into one large cavity.

If, however, the mastoid cells are involved, it will be necessary to do the more radical operation, which has been called the tympano-mastoid exenteration. The name explains the operation. Every particle of carious bone is removed from the mastoid and the posterior canal wall and scutum; the membrane and ossicles, if present, are removed and this region thrown into one large cavity. The wound behind the auricle is generally closed at the time of the operation and drainage carried out through the canal.

The following cases will serve to illustrate the points of diagnosis mentioned:

Case I.—I. R. M., aged five years. In this case there had been a discharge from both ears for more than two years following an attack of scarlet fever. He had been treated with fair regularity by different physicians, and had his adenoids removed. When first seen there were large masses of granulation tissue in both tympani; the discharge was profuse, but no exposed bone could be detected, and there was no mastoid tenderness. During a two months' course of treatment the granulations were gotten rid of, the discharge had greatly diminished, and the patient was apparently improving; but at the end of two months he missed a few visits, having been placed under the care of his family physician for general treatment owing to a sudden failure of health and strength. As there was no improvement he was seen in consultation. There was very little discharge from the left ear and none from the right. The upper and posterior canal wall of the right ear was red and tender, but not much swollen. Temperature 104° F. Some tenderness over the right mastoid, but not marked. A saline laxative was ordered and hot douching started at once and pushed vigorously. Twenty-four hours later the temperature was 100° F.; there was some discharge from the right ear and an increase from that in the left. Tenderness over the mastoid had disappeared, and the canal wall was less red, tender, and swollen. This apparent improvement continued for forty-eight hours, when his temperature went up to 105° F.; there was again some tenderness over the mastoid and the canal wall was more swollen and tender. It was decided to operate at once. A tympanomastoid exenteration was done; the mastoid found to be full of pus, the dura exposed in two places and the lateral sinus uncovered. An unfortunate accident occurred, but I do not believe it affected the result. While removing the canal wall a ledge of bone very unexpectedly gave way and the spoon shot across the mastoid cavity and opened the sinus; the bleeding was very profuse, but easily controlled by pressure.

The temperature dropped to 99° F. following the operation, but within fourteen hours reached 104° F. again, and fluctuated between 100° F. and 104° F. for seven days, when, in consultation with Dr. Randall, it was decided to explore the sinus for thrombosis. The wound in the sinus wall was found to be healed and the sinus itself free from thrombus; the jugular was exposed and it, too, found free. The temperature remained high and the patient gradually sank until forty-eight hours after the second operation, when he died.

The opening of the sinus was unfortunate, but not necessarily fatal, as proven by the fact that it occurred in another case of the writer's, in which necrosis extended so deeply that the bulb of the jugular was nicked; the patient made a complete recovery. The same accident has happened to other operators a number of times without serious consequences other than the delay in the operation to check the bleeding. Pyæmia was doubtless the cause of death in this case, and the question suggested itself whether it would have been better to have opened the left mastoid as well as the right at the time of the first operation, even though no symptoms pointed to that side; it is doubtful if this would have saved the patient's life. An operation two months earlier would probably have given a different result; but except for the profuse discharge then there was no sign of mastoid involvement.

Case II.—C. M. T., aged eight years. Discharge from left ear since she was a baby. Three days before operation, without any history of pain, a swelling appeared over the mastoid coincidently with the cessation of discharge; twenty-four hours later the pus again began to flow freely from the canal and the swelling over the mastoid disappeared. A tympanomastoid exenteration was done; the antrum and mastoid were full of granulations and pus, and the sinus was uncovered by caries of its wall.

Here we have a swelling over the mastoid that disappeared at the end of forty-eight hours; but the condition found at the operation shows what a great mistake would have been made in considering this case improved simply because the swelling had disappeared.

Case III.—M. B., aged twenty-six years. Profuse discharge and some odor from right ear for four years following a cold. No history of pain at any time, but firm pressure elicited tenderness over the antrum. There was a perforation in the upper posterior quadrant of the membrane. A six months' course of treatment failed to give any relief from discharge, and as some tenderness was present at times an operation was advised. The Stacke operation was performed and the antrum found to be full of granulations. Six weeks after the operation the discharge has ceased. In this case the profuse discharge and the occasional tenderness was suggestive of antral trouble and the operation confirmed the opinion.

Case IV.—A. F., aged thirty years. Discharge from right ear for six years. Four months ago began to complain of pain behind the ear. Examination showed marked tenderness over the antrum; discharge scanty; some bulging in the anterior flaccid membrane with a pulsating perforation. Free paracentesis through the bulging membrane was done at once, and this followed by hot douching and saline laxatives. Relief followed this treatment immediately; but the symptoms recurred four times within five weeks and operation was advised. The tympanic exenteration was done and the antrum found to be full

of granulations. After several weeks' treatment the case was discharged cured. She was seen two years later for impacted cerumen of the left ear; there has been no further trouble in the right ear.

Case V.—E. B., aged sixteen years. Discharge from right ear since childhood. Has had three abscesses over the mastoid which were opened and then healed. Now presents a small sinus behind the auricle about 5 mm. in width, from which there is a very slight oozing. Membrane entirely destroyed; no tenderness at all; temperature normal. The tympanomastoid exenteration was done. The mastoid cells were obliterated to a large extent by the sclerosis that is generally present in these chronic cases, and through this hard bone a sinus passed, in a gradually widening track, to the antrum. The latter was full of granulations and pus. Healing was slow in this case, but complete at the end of four months.

Case VI.—C. R., aged twenty-seven years. Discharge from left ear since childhood. Six times during the past ten years there has been a swelling over the mastoid, which opened spontaneously and was allowed to close. When first seen there had been no swelling for eight months and the soft tissues were completely healed. No pain or tenderness except at times of the swelling. There was a medium-sized perforation of the membrane up and posteriorly. Careful treatment for the suppurative otitis media was instituted and the patient watched. At the end of six weeks the discharge ceased and the ear was apparently in good condition; but on account of the history the patient was told to return every few weeks. The ear remained quiet for three months; then the patient reported with the history of pain and discharge two days previously. The canal was full of cholesteatomatous masses. The membrane was intact, the old perforation being closed by a depressed cicatrix. On the upper and posterior canal wall was a mass of granulations and through this mass bare bone and what felt like a sinus could be detected. Temperature was normal. A tympanomastoid exenteration was done. Dense cicatricial tissue, tightly bound down to the bone, was found over the mastoid; the bone to the depth of about 5 mm. was almost ivory-like in hardness, but from that point there was a cavity filled with pus and granulations. An opening through the tegmen exposed the dura and a sinus was found leading from the mastoid to the posterior wall. As in Case V., the healing was slow, requiring about five months, but it has been complete.

Cases V. and VI. show that the presence of a sinus leading into the mastoid, whatever its location, is a sufficient indication for operation, and they also serve to illustrate the inefficiency of the Wilde incision for any case of true mastoiditis. In Case V. the abscess was opened with a knife, in Case VI. it opened spontaneously; but the result was practically the same in each case.

Case VII.—J. S., aged forty years. Discharge from right ear for thirty-three years following scarlet fever. During the past fourteen weeks has been having attacks of vertigo, so called. The attacks would appear at any time during the day or night and with increasing frequency, so that he might have five within twenty-four hours; he became very dull, stupid, and irritable. The tympanic membrane was

intact, but showed a depressed cicatrix up and posteriorly. Lying loose on the floor of the canal was a sequestrum measuring 15 x 5 x 5 mm., which had come through a sinus in the upper posterior canal wall and through this sinus the pus was flowing and showing a distinct pulsation. There was no tenderness over the mastoid and no swelling. Temperature 99° F. A tympanomastoid exenteration was done; in this case also the outer bone for about 4 mm. was extremely hard, but beyond this it was all one cavity. A small area of the tegmen was destroyed, exposing the dura; and posteriorly there was an extradural abscess about 25 x 35 mm.

Healing in this case was slow, requiring about five months, but it was perfect. Following the operation there were no epileptiform attacks for three weeks, but at that time one occurred; this was accompanied by a partial palsy of the right side of the face. Nothing of a suspicious nature was found about the site of the operation, and as the palsy cleared up entirely within three days it was probably due to the fact that the gauze was packed into the wound too tightly. There were four of the epileptiform attacks during the three months following the operation, but a course of bromides controlled them completely. The patient was seen fifteen months after the operation and was feeling bright and well and had gone back to his work, that of a day laborer.

The sinus, sequestrum, and epileptiform attacks, indicating the probability of cerebral pressure, presented an unmistakable and urgent indication for operation. This patient resembled Case VI. in the closing of the perforation of the membrane and the formation of a sinus through the canal wall.

A brief review of the symptoms indicating operation in many cases of chronic suppurative otitis media would then be this:

Profuse and long-continued discharge suggestive, but not diagnostic, of antrum or mastoid involvement.

The fluctuating swelling over the mastoid diagnostic when present, but its absence not contraindicating operation. Œdema behind the auricle is more apt to be due to furuncle. Tenderness over the mastoid, especially in the region of the antrum, of great significance whenever present, especially if it is persistent; but this may be doubtful or absent if the outer table is much sclerosed.

The inflamed, tender, and bulging upper and posterior canal wall, one of the most important signs of all, and its non-relief by treatment is a sure indication for operation. A sinus, whatever its location, leading into the mastoid is a positive indication.

Facial palsy of interest, but not important.

Epileptiform attacks unusual, but indicating possible cerebral pressure.

Marked and rapid failure of health, more apt to be found in adults than in children.

The temperature more apt to be elevated in children than in adults.

The rapid fluctuation of temperature through a range of several degrees our most important sign in cases of pyæmia.

The slow and thready pulse, out of proportion to the elevation of temperature, irregularity or sluggishness of pupils, rapid failure of health, rigors or convulsions, signs of intracranial pressure.

Tenderness and œdema over the occipital are very apt to be present when the sinus is thrombosed. Bacteriological examinations not positive, but important.

A XIPHOSTERNAL CRUNCHING SOUND.*

WITH A REPORT OF SIX CASES.

By Myer Solis-Cohen, A.B., M.D.,
ASSISTANT PHYSICIAN TO THE MEDICAL DISPENSARY OF THE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA, ETC.

As assistant to Professor M. Howard Fussell, Chief Physician to the Medical Dispensary of the Hospital of the University of Pennsylvania, I examine about one-fourth of the patients applying to that service. Of 492 cases examined by me during a period of fifteen months, I observed in six (or 1½ per cent.) what I have termed a xiphosternal crunching sound. This sound is superficial in character, and may be compared to the sound caused by a boot treading on soft snow. It is heard over the lower end of the sternum and a little to the left of it. The exact area over which it is heard varies somewhat with each case. The upper margin was between the upper border of the fifth rib and the upper border of the sixth rib. The lower boundary ranged from a line drawn transversely through the base of the ensiform to one extending from the tip of the xiphoid to the junction of the seventh costal cartilage with the costal margin. The right border in every case lay between the right and left edges of the sternum. The left boundary was between the left mid-clavicular line and a line drawn an inch and a half to the left of the sternum and parallel with it. The sound was heard during the whole of the cardiac systole, but not during diastole. It varied in intensity, and occasionally slightly in position, but never in character. It was not influenced by exercise, respiration, or pressure with the stethoscope, except in one instance, in which it seemed to be increased when the stethoscope was pressed against the chest, and also when the patient had walked rapidly. When the patient leaned forward the sound became intensified (in every case but one), and when he lay down it diminished in intensity, in two cases completely disappearing. In every case the heart was enlarged, and the cardiac sounds

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were weak and of poor quality. Retraction of the interspaces was noted in three cases. The pulse averaged about 82.

This sound is not described in any text-book on general medicine that I have examined, although I have searched the works of Allbutt, Anders, Barlow, Bartholow, Bennett, Bristowe, Chapman, Davis, Eichhorst, Fagge, Flint, Gibson, Gregory, Lockwood, Loomis, Lyman, Mackintosh, Money, Osler, Pepper, Reynold, Roberts, Roger, Stillé, Strümpell, Tanner, Taylor, Loomis-Thompson, Tyson, Watson, and Wood and Fitz. I have found no mention of it in any of the following standard text-books on diagnosis: Coiffier, Corwin, Da Costa, Debove and Achard, Edlefsen, Gibson and Russell, Hare, Hutchinson and Rainy, Jacob, Klemperer, Kuhnemann, v. Leube, Musser, Tyson, and Vierordt. Mayet,[1] however, speaks of a xiphoidal exocardial or cardiopulmonary murmur resulting from the action of the heart against the lung. Butler[2] speaks of a "friction sound heard over the lower end of the sternum and the adjoining costal cartilages, synchronous with the action of the heart," which, he thinks, "may be due to subphrenic peritonitis or abscess, or perhaps to a diaphragmatic pleuritis."

In their works on the heart no sound in any way resembling the xiphosternal crunching sound is mentioned by Barth and Roger, Balfour, Bellingham, Bertin, Billing, Blakiston, Broadbent, Bramwell, Clark, Da Costa, Flint, Fothergill, Gerhard, Gibson, Hayden, Hope, Keating and Edwards, Latham, Paul, Shapter, Walshe, Wardrop, Waters, or Watson.

In a careful review of the periodic literature treating of the heart and pericardium, I find very few references to a sound in any way resembling that forming the subject of this paper.

In 1826 Laennec,[3] in speaking of certain phenomena that might be confounded with true cardiac murmurs, described a "metallic click heard over the præcordial region in subjects with nervous palpitation," which he believed to be produced by the impulse of the heart against the thoracic parietes. He also mentioned having observed in other cases in the same region a sound resembling "the creak of leather of a new saddle under the rider," which at one time he regarded as a sign of pericarditis, but later became convinced that it was not. It seemed to him to occur when the heart, enlarged or distended with blood, was crowded in the inferior mediastinum, as if there were bubbles of air in the pericardium.

Bouillaud,[4] in 1835, and Aran,[5] in 1843, referred to "the metallic tinkling sound" pointed out by Laennec, describing it as "a metallic clacking of more or less intensity, of which an idea may be formed by applying the palm of the hand on the ear and tapping lightly the back of this hand with the finger of the opposite hand."

William Stokes,[6] in 1855, mentioned a case of probably fatty degen-

eration of the heart, in which he found over the right ventricle a rasping sound, which disappeared in a few days, after the application of a blister and the use of a few mercurial pills.

"Xiphisternal or Pericardial Chisel-sound, with its Practical Application," was the subject of a paper read in 1856 by F. J. Brown,[1] in which he stated that "a chisel-sound at the lower end of the sternum is of frequent occurrence in persons that are in good health in all respects excepting dyspepsia. . . . The sound is that of a chisel or short plane used forcibly across the end of a piece of timber. It is single and synchronous with the systole. It varies in intensity, but never in character. . . . It is exocardial.

"The boundaries of the cardiac-percussion sound are extended more or less, oftentimes but slightly.

"The symptoms that commonly coexist with xiphisternal chisel-sound are those of a form of dyspepsia.

"The treatment of xiphisternal chisel-sound consists in the use of mild antiphlogistics and resolvents and in the local abstraction of blood, followed by counterirritations."

Twenty cases are reported in this article, some with complications, including cardiac hypertrophy (four cases), degeneration of the heart (one case), aortic disease (one case), rheumatism (two cases), ecthyma and chorea (one case), hypochondriasis (two cases), ovarian disorder (two cases), cerebral affection (two cases), and epistaxis and splenalgia (one case).

Brown is of the opinion that "the sound is due to 'white pericardial patches' plus toxæmia," and that "the attendant dyspepsia is only sympathetic."

In 1882 Angel Money[2] published the result of a series of very careful observations made on the hearts of 111 parturient and puerperal women who were admitted to a lying-in hospital over a period of six months. In 84 of these, or, roughly, 75 per cent., murmurs were heard, which were divided by Money into three sorts.

The "second sort (friction-like) was systolic in time, was almost absolutely not conducted, being heard only over a very small area to the left of the median line of the sternum. It sounded quite superficial, and was short and high-pitched and rather stiff in quality. This friction-like systolic murmur was to be heard just above and to the left of the ensiform cartilage. It was usually audible only over an area which might be covered by the small end (of moderate size) of the stethoscope. . . . The seat of the murmur would about correspond to the 'white patch' so often found on the right ventricle. I have no post-mortem evidence to offer on this point. My experience enables me to say that in all cases this murmur was a temporary phenomenon. The bruit was detected in 29 cases out of 111. It was the sole murmur in 22. It was associated

six times with a murmur of the first sort; twice with one of the third sort.

"I have stated my belief that the murmur is in some way related to a rubbing of the visceral over the parietal lamina of the pericardium, which laminæ may or may not be altered in some degree in structure."

Under the heading of "Rough, Scraping, Systolic Sounds Heard Over the Tricuspid Area and the Right Ventricle," A. E. Sansom,[9] in his text-book published in 1892, takes issue with Money as to the origin of the sound described by the latter. He says:

"There can be no doubt that short, rough, systolic, seemingly very superficial sounds are often to be heard over the situation of the right ventricle in conditions which deviate in no notable way from the standard of health, and these may be very difficult (or impossible) of explanation. It is very improbable that these are due to 'milk spots,' or to any change in the pericardium, visceral or parietal. Such supposed thickenings over the pericardium are smooth and polished, and unlikely to give the peculiarly rough sound observed."

E. H. Colbeck,[10] in his work on *Diseases of the Heart*, published in 1901, says:

"A peculiar form of friction sound is sometimes heard over the right ventricle at the level of the fifth and sixth intercostal spaces, close to the left sternal edge and over the base of the ensiform cartilage. It is possibly associated with the formation of the 'white patch,' which may be observed on the underlying portion of the pericardium covering the heart. The sound is systolic in its relation to the cardiac cycle, and may at one time roughly resemble a reduplication of the first sound; at another, an endocardial bruit. It may vary in character between a faint click and a definite rub, and, as a rule, the intensity of the sound is greatly modified by changes in the position of the body. The sound is most commonly observed in downward displacement of the heart, due to emphysema, but it may be present under apparently normal conditions."

"An Undescribed Cardiac Sound" was the name applied by H. A. Hare[11] in 1901 to a "peculiar vibrating sound occurring with systole, and heard best in an area extending from one inch to the right of the sternum to one inch to the left of the nipple, on the nipple level, and which is sometimes so dry, if I may use such a term, that I have at times considered it somewhat like the pericardial friction sound heard in pericarditis near the base. That it is not a friction sound I am very confident, although I must admit that its cause is not clear. Personally I believe it to be due to vibrations in the chordæ tendinæ produced by imperfect contraction of their muscular attachments, because I have never met with the sound except in cases of general debility with a feeble irritable heart. . . . . The sound I refer to is not a murmur in

the ordinary sense, but a vibratory sound resembling the sound 'ching' at each systole. It is short and quick, not changed by deep or superficial pressure of stethoscope, and is not entirely put aside by rest in many cases, although it is usually decreased by this means. It becomes accentuated by nervousness or fear, but not by exercise of a sufficiently violent kind to distinctly accentuate ordinary sounds."

Osler[12] in discussing Hare's paper said:

"There is a distinct difference in the majority of healthy hearts between the first sound over the apex and that as you move toward the sternum, a more crunching sound, which is peculiar and remarkable."

I called M. Howard Fussell's attention to the xiphosternal crunching sound exhibited by each of the six cases I am reporting. Dr. Fussell (1902, personal communication) in each case identified the phenomenon with a crunching sound frequently observed by him in cases of myocarditis, of neurasthenia, and in some cases of anæmia; and he was of the opinion that the sound was produced in the cardiac muscle.

The histories of the six cases I have observed are appended.

The patients were all men, five white and one colored. The ages ranged from eighteen to sixty-three. All but one were married. The occupations included both light and heavy work.

The *family history* was negative except in one case, where the patient's mother had rheumatism and his brother had died of dropsy.

Two of the patients had indulged in alcohol to excess and two had not touched it at all. Three were addicted to excessive use of tobacco; one never employed it.

Inquiry into the *previous medical history* showed that the following diseases and symptoms had been complained of by the number of cases recorded:

Typhoid fever, 2; pleurisy, 1; rheumatism, 4; urticaria, 1; gonorrhœa, 4; syphilis, 2; coryza, 3; sore-throat, 4; influenza, 4; hemorrhoids, 3; cough, 4; expectoration, 3; night-sweats, 2; loss of flesh and weight, 2; dyspepsia, 3; præcordial pain, 2; dyspnœa, 4; palpitation, 2; vertigo, 4; weakness, 5; œdema, 3; headache, 2.

The *symptoms* complained of by the patients on admission, and during the course of their illness, follow:

Anorexia, 5; malaise, 4; weakness, 3; headache, 2; dyspnœa, 3; palpitation, 4; vertigo, 2; pain about the heart, 4; general soreness, 2; tenderness and soreness of the extremities, 3; pain in the shoulders and back, 4; nausea, 2; heartburn, 4; pain in the stomach, 4; abdominal pain, 4; eructation, 2; constipation, 2; diarrhœa, 2; drowsiness, 3; distention of abdomen, 1; hemorrhoids, 3; expectoration, 5; sore-throat, 2; nasal trouble, 3; nose-bleed, 2; loss of weight, 1; headache, 3; seminal emissions, 4; oligoria, 2; nervousness, 1; urticaria, 1.

With two exceptions the patients *on admission* were well developed and well nourished. The pupillary reactions were normal. In four cases the tongue was coated. The pulse was regular in every case, weak in three, of low tension in two, and compressible in three. The rate varied from 60 to 104, the average being about 82. In four instances the radial arteries were fibrous. Pulsation was visible in the neck in two cases, below the clavicle in one, in the epigastrium in one, and at the apex in three. Expansion was diminished in two of the patients. Retraction was noted in the intercostal spaces in three cases, and at the epigastrium in two. One patient exhibited capillary engorgement; another, cyanosis. In every case the heart was enlarged, and with one exception, the cardiac sounds were weak and of poor quality. The lungs were negative in every case but one.

The *blood* count was practically normal. The hæmoglobin averaged about 85 per cent. The coagulation time was about two minutes.

The *sputum* in every case was negative.

Albumin was present in the *urine* in three cases; casts in two.

The examination of the *stomach contents* was made in two cases and a condition of subacidity found to be present.

The xiphosternal crunching sound, heard in every case, is a sound difficult to describe. In my notes taken before I decided upon this name, I employed various terms in describing it, using such expressions as "sounds of harsh clicking or rubbing character and high-pitched," "friction sound," "rough rubbing, clicking sound," "brushing sound," "sound resembling the rubbing together of two pieces of velvet," "scraping sound," and "that peculiar, gentle, rubbing sound."

The sound undoubtedly does vary somewhat; during an examination lasting fifteen minutes, it may be scarcely perceptible at one time, while quite loud and distinct at another. In only one case did it disappear entirely, and then but for a short time. I have been unable to discover to what influences the variations are due.

Notwithstanding the difficulty with which the sound is described, after it has once been heard it can instantly be recognized when observed again. I consequently believe that the sound heard in each of the six cases was of one and the same character.

But I cannot say that this is the sound heard by the authors I have quoted. I do not think that this is the sound referred to by Mayet, by Butler, by Bouillaud, or by Aran. It may have been the one compared by Laennec to the "creaking of leather," but probably was not that described as "a metallic click." It is my opinion, however, that the xiphosternal crunching sound heard by me is identical with the sounds described by Stokes, Brown, Money, Sansom, Colbeck, Hare, and Fussell.

The cause of this sound can only be a matter of conjecture until a sufficiently large number of autopsies have been made on cases which

have at one time exhibited this phenomenon. It is quite possible that the sound is produced in several ways. The sound that disappears under mild antiphlogistic treatment and the one that persists in spite of treatment may have different origins.

I do not think that the sound heard in my cases was caused by the impact of an enlarged heart on the lingula of the lungs, or by the friction-rub of pleuropericarditis. I incline to the view that it was due to the presence of white patches on the pericardium. This is not so irrational a supposition as some writers seem to believe.

In Allbutt's *System of Medicine*, F. F. Roberts[13] says:

"From personal observation, however, I feel sure that some white spots or patches on the pericardium are capable of originating a limited friction sound."

Gibson,[14] in speaking of pericarditis, says:

"Although the friction sound is almost pathognomonic, it is nevertheless sometimes, although not often, found in the absence of pericarditis. . . . Milk spots upon the pericardium, dryness of its surface, and ecchymoses into the subserous tissues have long been recognized as causes."

It is well known that white patches may be present on the pericardium without giving rise to a friction or crunching sound. It is quite possible that they cause this phenomenon only when associated with certain conditions of debility or of myocardial weakness. It seems also possible that the gastric irritation present in all of Brown's cases, and in five of mine, may have been due to a passive congestion of the gastric mucous membrane, secondary to the myocardial weakness.

Until more cases are reported of this fairly common condition, it will be impossible to form any conclusions; and until post-mortem examinations with this phenomenon in mind are made upon bodies that during life exhibited the xiphosternal crunching sound, nothing definite can be stated about its origin.

Report of Cases.

Case I.—H. W., male, aged twenty-eight years, a laborer, was admitted on March 13, 1902. The family history was negative. The patient had had measles, scarlet fever, influenza, hemorrhoids, a slight morning cough, with the expectoration of thick, tenacious sputum; dyspnœa, which was worse on exertion; and, more recently, night-sweats, loss of flesh, weight, and strength, and vertigo.

For the past two months he had suffered with soreness all over, but most marked in the chest and legs, anorexia, and malaise. Several times a day he experienced nausea, which was worse on exertion. He often had heartburn. His feet did not swell.

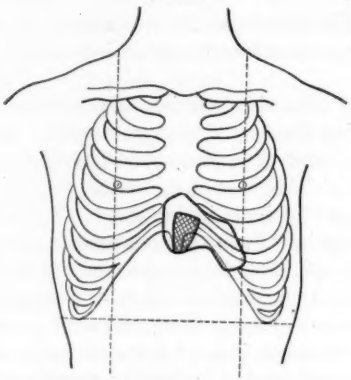
He was a well-developed but poorly-nourished man, with rather pale skin. His pupils were equal and reacted well to light and accommodation. His pulse was 84, regular, rather weak, and of low tension.

The radial arteries were somewhat fibrous. The palms were moist. The tongue had a dirty yellow coating on the back part. The temperature was 98.8°. The chest and abdomen were of fair development. The movement of the chest seemed labored and rather restricted, especially on the right side. There was pulsation in the neck and below the clavicles. A fluttering pulsation was visible in the sixth interspace, in the left mid-clavicular line. There was slight bulging of the left chest with increased consistence, it being much firmer above the left clavicle than above the right.

The apex beat was not palpable. The heart was enlarged to the left. The sounds were heard best in the sixth interspace, the first and second sounds here being similar in character. The sounds were weak over the body of the heart and at the base.

At the ensiform, and above and to the right of the apex, a harsh sound of a clicking or rubbing character, and high-pitched, was heard, synchronous with the heart sound, not increased by movement, and not noted when the patient was lying down.

FIG. 1.



The lungs presented no abnormality.

The abdomen was tender, but otherwise normal.

The extremities were normal.

The urine contained neither albumin nor sugar.

Examination of the sputum was negative.

The blood on examination showed: hæmoglobin, 92 per cent.; red blood cells, 5,000,000; leucocytes, 7440. The stained specimen was normal.

The patient improved under stomachic treatment.

The only symptoms of note while the patient was under observation were sore-throat, nose-bleed, soreness of scalp, and a papular eruption.

The peculiar cardiac sound was heard more plainly on March 17th; on April 5th it was increased by pressure with the stethoscope and after the patient walked rapidly, and did not disappear when he lay down; on April 11th it could not be heard at all; but on April 16th and on May 3d it was plainly audible.

The pulse on subsequent examinations was 76, 96, 80, and 80.

CASE II.—W. O., male, white, aged eighteen years, single, an upholsterer, was admitted on June 25, 1902. The family history and the past medical history were negative.

On admission, the patient complained of a constant though not severe pain in his stomach of a week's duration, at times relieved by eating, to return again in about an hour; of anorexia, malaise, and eructation.

The patient was a well-developed and well-nourished man. His pupils were equal and reacted normally. His tongue was clean. His pulse was 104, regular, of fair strength and tension, and compressible. The hands were somewhat cyanotic.

His chest was well developed, the right side being more prominent (a condition since birth). The sternum was depressed at its lower end. There was no epigastric pulsation. No retraction was visible anywhere.

Expansion was fair, though greater on the right than on the left.

A diffuse impulse was noted in the fifth interspace, left mid-clavicular line, and radiating to the fourth interspace above and to the left for several inches. The apex beat was distinctly felt in the fifth interspace, just inside the mid-clavicular line. The heart was enlarged to the right. . The heart sounds were weak and lacking in muscular quality.

Over an area bounded, above, by the top of the fifth rib; below, by the lower edge of the sixth rib; on the right, by the mid-sternum; on the left, by a line an inch and a half to the left of the left sternal border, a rough, rubbing, clicking, brushing sound was heard, superficial in character. The sound was unchanged during inspiration or expiration, or when the breath was held at the end of inspiration or expiration. It was not increased by exercise, by pressure with the stethoscope, or by the patient leaning forward, but it was somewhat diminished when he lay down. The sound varied in intensity, but according to no discernible rule. The area over which the sound was heard always remained the same.

The lungs were normal.

There was no œdema of the legs. The knee-jerks were diminished.

The abdomen was normal.

The urine was acid in reaction and contained a slight trace of albumin, but no sugar. The microscope showed only bacteria.

The patient was put on stomachic treatment and at once began to improve.

The presence of the peculiar cardiac sound was noted on July 15th, and December 9th.

CASE III.—C. S., white, aged sixty-three years, married, a carpenter by trade, was admitted on July 3, 1902. The family history was negative. The patient had had typhoid fever, influenza, gonorrhœa, and hemorrhoids, and had suffered from recurring pains across the back, dyspnœa on exertion, and occasional vertigo.

About a month before he applied to the dispensary, the patient had "caught cold." For the last two weeks he had had an uncomfortable feeling in his stomach, not influenced by his meals. He had felt some nausea, but had not vomited. On admission he complained of constant pain in his back over the right sacral region, at times leaving the back and going to the arms, the left lumbar or the umbilical regions. The patient had lost ten pounds in one month. His appetite was rather poor.

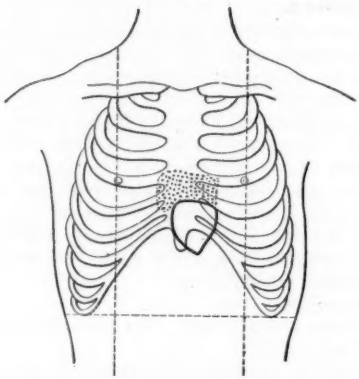
The patient was a thin man, having a somewhat shrivelled appearance. His pupils were normal. The tongue was coated. The pulse was 60,

regular, weak, of good tension, and not compressible. The radials were fibrous, but the temporals were not. There was some cyanosis of the hands.

Retraction during systole was noted in the region of the ensiform between the two costal margins, being more marked in the space between the xiphoid cartilage and the left costal border. It was increased on inspiration and greatly diminished on expiration. There was no movement of the ensiform itself.

The apex beat was neither visible nor palpable. The heart was enlarged to the left. The heart sounds were weak and were lacking in muscular quality. They were heard best in the sixth interspace, half an inch to the left of the mid-clavicular line, and were scarcely audible at the aortic cartilage and at the pulmonary area. A loud sound was heard in the region of the ensiform, being louder and most distinct over an area the size of a silver dollar,

FIG. 2.



bounded by the seventh rib, above; the tip of the ensiform, below; the middle of the xiphoid, on the right, and a line half an inch to the left of the costal margin, on the left. It was louder on inspiration and became much fainter when the patient was lying down. The sounds in the vessels of the neck were very faint. A rubbing sound, resembling the rubbing together of pieces of velvet, was heard over an area bounded: above, by the fifth rib; on the right, by the right sternal border; on the left, by a line beginning an inch and three-quarters to the left of the left sternal border and extending to the mid-clavicular line; and below by a line extending to the tip of the ensiform across to the left costal border, and then along the eighth costal cartilage. The sound was heard best over this area, but was transmitted a short distance. It did not seem to be affected by respiration, apparently being equally loud during inspiration and expiration, and when the breath was held at the end of either movement. Sometimes, however, it was noted louder during inspiration. It was not influenced by pressure with the stethoscope. It was a little louder when the patient leaned forward, and when he lay down it became much fainter.

The lungs and abdomen were normal.

The quantity of urine passed in the twenty-four hours was twenty-six ounces. The specific gravity was 1040. The urine was dark amber in color, clear, and acid in reaction. A trace of albumin was found, but no sugar. Under the microscope were seen hyaline, coarsely granular, and epithelial casts; cylindroids; leucocytes; renal epithelium, and crystals of uric acid and of calcium oxalate.

The sputum contained leucocytes and diplococci, but no tubercle bacilli.

The blood contained 68 per cent. of hæmoglobin.

The patient improved under glonoin and Basham's mixture.

On November 25th and on December 11th the peculiar sound was still heard.

CASE IV.—S. W., white, an Hungarian, aged twenty-five years, married, a weaver by trade, was admitted on September 29, 1902, complaining chiefly of pains in his back, chest, arms, and legs. The family history was negative. The patient had had typhoid fever, urticaria, and occasionally sore-throat. For the past few years he had suffered with slight dyspnœa and palpitation on exertion, continuous headache, and loss of flesh, weight, and strength.

The illness for which the patient applied had begun four months previous, with "a cold in the stomach," as the patient termed it, and with a diminution in the amount of urine passed. For a month or so past he had suffered from a burning sensation in the epigastrium and left hypochondrium after both dinner and supper, lasting three or four hours and accompanied by eructation. On admission, the patient complained of anorexia, malaise, and epigastric tenderness; of pains in his chest, back, arms, and legs, which were worse when the weather was bad; of dyspnœa and palpitation on exertion; of vertigo, and of weakness.

The patient was a well-developed, though poorly-nourished man. His conjunctiva, lips, and gums were pale. The left pupil was slightly larger than the right, but both pupils reacted normally to light and distance. The tongue was coated; the pulse was 72, regular, of fair strength and tension, and not compressible. The radials were not fibrous.

Expansion of the chest was good. In the fifth interspace, half an inch inside the mid-clavicular line, there was seen a slight fluttering, appearing as an impulse when the patient leaned forward, but more as a retraction when the patient stood erect. No other pulsations or retractions were visible.

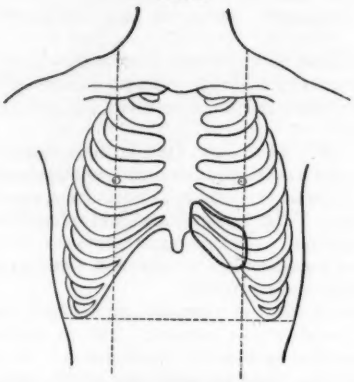
The apex beat was not palpable. The heart was enlarged to the right. The heart sounds were weak and of poor quality, and were heard best in the fifth interspace in the mid-clavicular line. Over an area the size of a silver dollar, bounded above by the lower border of the fifth rib, below by the junction of the seventh rib with the sternum, on the right by the right sternal border, and on the left by a line one inch to the left of the left sternal border, was heard a rough and brushing, or rubbing or scraping sound, occurring during systole; observed during inspiration and expiration, and when the breath was held at the end of inspiration or expiration, but heard best near the end of expiration, and not influenced by pressure with the stethoscope or by exercise, When the patient leaned forward the sound was increased in intensity, and was heard over a larger area, which extended upward to the fourth

rib, and on the right to a little to the right of the right sternal border. When the patient was lying down the sound was not heard.

The lungs were resonant and the breath sounds were normal.

The abdomen was normal and the abdominal organs were normal. The boundaries of the stomach (inflated by means of a hand-ball syringe

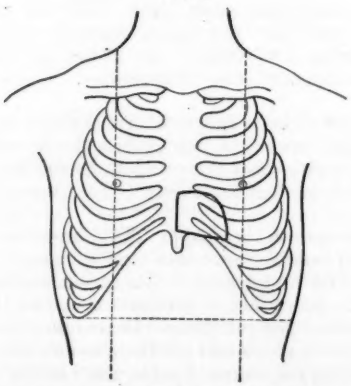
FIG. 3.



attached to a stomach tube) were as follows: upper, fifth rib; lower, eighth interspace; left, a quarter of an inch to the left of the mid-clavicular line; right, the right costal border.

The patellar and plantar reflexes were absent.

FIG. 4.



The urine was amber in color, turbid, acid in reaction, and its specific gravity was 1025. It contained a trace of albumin, but no sugar. Under the microscope a few hyaline casts were seen; also, a few round, columnar, and squamous epithelial cells, and amorphous urates.

The sputum contained leucocytes, but no tubercle bacilli or any other micro-organisms.

Examination of the blood resulted as follows: hæmoglobin, 91 per cent.; red blood cells, 4,000,000; leucocytes, 14,800; coagulation time, two minutes. The red blood cells stained very faintly, but showed no degenerative changes. There were a very few microcytes, megalocytes, and poikilocytes. The different forms of the leucocytes were present in the following proportions: polynuclear neutrophiles, 57 per cent.; large mononuclear basophiles, 4 per cent.; small lymphocytes, 32 per cent.; eosinophiles, 7 per cent.

The stomach contents were examined two hours after an Ewald test breakfast had been ingested; 11 c.c. were expressed of a brown, chocolate-colored fluid, thick with undigested particles. Under the microscope starch-granules and a granular detritus of undigested matter were seen. The reaction was acid. Free acids and free hydrochloric acid were present. There was no trace of lactic acid. 0.1 c.c. of a decinormal solution of sodium hydrate neutralized 1 c.c. of the filtrate.

The patient was put on the treatment advised by Dr. Brown (*loc. cit.*), consisting of pills of tartar emetic and extract of hyoscyamus, and of a mixture containing liquor ammoniæ acetatis and potassium iodide, which caused such nausea and vomiting, however, that it had to be discontinued.

The xiphosternal crunching sound was heard on subsequent examinations. The pulse at different times registered 72 and 88.

Case V.—G. P., colored, aged forty-three years, married, a laborer, was admitted on October 3, 1902. The family history was negative. The patient denied having contracted syphilis. He had had the diseases of childhood, rheumatism, malaria, influenza, pleurisy, and hemorrhoids. He had experienced heartburn. Recently he had been suffering from continuous headache, malaise, vertigo, and weakness. There was no family history of rheumatism, of heart, lung, or kidney disease, or of dropsy.

About a week before admission the patient had been thoroughly drenched about the legs and feet, and since then he had had general pains, and also severe pain in the breast, especially on the left side, so that he was unable to stoop on that side. He complained of dyspnœa and palpitation on the least exertion, but never otherwise. He was weak and unable to work.

The patient was an exceedingly well-developed and strong-looking man. A small swelling, adherent to the bone, but not to the skin, was present over the right frontal bone. His right eye had been "picked out by a crane," but the pupil of his left eye reacted normally. His tongue was coated and flabby. The pulse was 76, regular, fairly strong, of high tension, and compressible. The radials were somewhat thickened, but the temporals were not.

The breathing was of the abdominal type; the chest did not move. On forced respiration expansion was diminished. Pulsation was noted in the vessels of the neck, but not in the epigastrium. A diffuse retraction was seen in the sixth interspace in the neighborhood and to the left of the mid-clavicular line.

The heart was slightly enlarged. The cardiac sounds were fair, but not of good quality. They were heard best in the sixth interspace, just outside of the mid-clavicular line. During the whole of systole a peculiar brushing sound was heard over an irregular area, bounded: above, by the fifth rib; below, by the seventh costal cartilage; on the right, by a

line an eighth of an inch to the left of the right sternal border and by the left costal margin; and on the left by a line one inch to the right of the mid clavicular line. The sound did not seem to be superficial. It apparently did not accompany the second sound of the heart. It was not affected by respiration nor by the holding of the breath, at the end either of inspiration or expiration. When the patient leaned forward the sound was heard louder and over a larger area, extended farther to the right, to the left, and below. The sound was not much affected by decubitus, but it might have become a little less loud, and the area might have become slightly diminished in size. The sound was intensified by exercise.

The lungs were apparently normal.

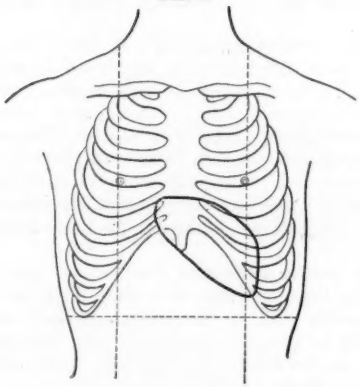
The abdomen was normal. The liver and spleen were not enlarged.

The legs exhibited no œdema.

The blood contained 94 per cent. of hæmoglobin and 4,700,000 red blood cells. The coagulation time was two minutes.

The patient improved under potassium iodide in increasing doses.

FIG. 5.



The xiphosternal sound at a subsequent examination was described as "a peculiar, gentle, rubbing sound."

CASE VI.—J. F. O. B., white, aged forty-two years, married, a leverman, was admitted on November 14, 1902, complaining chiefly of the fact that he caught cold easily.

The patient's mother had rheumatism, and one brother had died of dropsy.

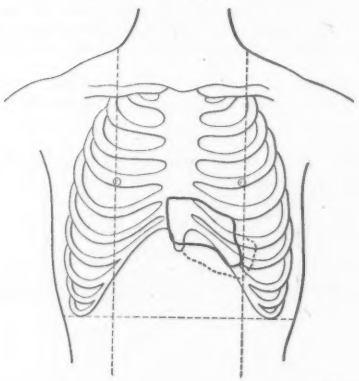
The patient had had measles, chicken-pox, urticaria, gonorrhœa, syphilis, malaria, lumbago, influenza, and eczema. For some years he had suffered from periodic headaches, occasional vertigo, winter cough, sore-throat and dyspnœa, and palpitation on exertion. He also experienced dyspeptic symptoms. He had aches and pains all over occasionally, especially six years ago. He had had lumbago several times.

The patient came referred from the throat and nose dispensary, saying that he caught cold easily. He suffered from itching about the rectum and from occasional epigastric pain and tenderness. He complained of weakness and of becoming easily tired. He said he had a fissure.

The patient was a thin, poorly-nourished man. His pupils were equal and reacted normally. His tongue was fairly clean, but roughened, furrowed, and slightly tremulous. His pulse was 92, regular, weak, and of fair tension. The radials were slightly fibrous.

The chest was emaciated and flat, and presented some capillary congestion. The superficial veins were dilated. The left scapula was prominent. Above and below the clavicles there were depressions on the right and some fulness on the left. The neck inclined to the right. There was a slight retraction in the sixth and seventh interspaces about the mid-clavicular line; also in the tenth and eleventh interspaces, in the region of the posterior axillary line and nearer the spine. A retraction during systole was noted between the ensiform and the left costal border, seen during inspiration—best at the end—and scarcely visible during expiration. Expansion was poor, being much restricted on the left.

FIG. 6.



The apex beat was neither visible nor palpable. The heart was enlarged to the left. The cardiac sounds were heard best in the sixth interspace, in the mid-clavicular line. The sounds were very weak and of poor quality at the apex; scarcely audible at the aortic cartilage, the first being particularly indistinct; also weak but somewhat louder at the pulmonary area, and louder still at the ensiform. A rubbing, harsh sound was heard to the left of the ensiform over a small area the size of a quarter, bounded roughly by the fifth rib above, the left border of the sternum and the left costal margin on the right, the mid-clavicular line on the left, and by the junction of the seventh rib with the costal margin below. The sound was very indistinct; sometimes louder at one time during a fifteen-minute examination than at another, and often not heard. At times it seemed to become louder when the patient leaned forward, but often it seemed little if at all affected by this position. It did not seem to be affected by pressure with the stethoscope. When the patient lay down this sound was not heard, and all the cardiac sounds were weaker. At times the sound was heard best during inspiration, sometimes just at the end, the breath being held; at other times at the end of expiration. It seemed to be heard better during the expiratory

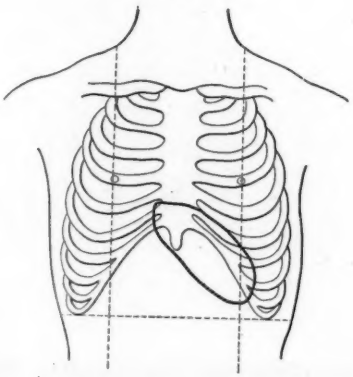
oftener than during the inspiratory act. The boundaries of the area were not very distinct. At no time was the sound very loud, and at the borders of the area at times it seemed to blend with the impure heart sounds heard over the præcordia. The sound accompanied the systole of the cardiac cycle. Exercise did not increase the loudness of this sound, but did increase the loudness of the ordinary heart sounds. No sounds were heard in the vessels of the neck.

There was dulness at the apex of the left lung anteriorly, and over the right chest posteriorly. The breath sounds were very faint. Expiration was possibly slightly prolonged on the right posteriorly. There were a few coarse râles on the right posteriorly.

Examination of the abdomen was negative. The outline of the stomach was as in the diagram. Two fistulæ in ano were present. The veins of the arms were distended.

Station and gait were both good. The patellar reflexes were normal. The plantar reflex was normal on the left, but very slight on the right.

FIG. 7.



No tubercle bacilli were found in the sputum.

The urine was of specific gravity 1010, acid, and contained no albumin or sugar.

The blood contained 80 per cent. of hæmoglobin, 4,800,000 red blood cells, and 5840 leucocytes. The coagulation time was between two and two and one-quarter minutes.

The stomach contents were extracted after a hearty meal. (The patient did not state that he had not taken the test breakfast ordered.) They were greenish-yellow in color, and contained undigested particles. The reaction was acid. No free acids were present; 0.4 c.c. of a decinormal solution of sodium hydrate neutralized 2 c.c. of the filtrate (or 20 c.c. neutralized 100 c.c.). There was no free hydrochloric acid and no lactic acid.

The only treatment given was a thirtieth of a grain of strychnine sulphate three times a day, the patient improving and feeling better than he had for years.

The xiphosternal sound was called, on November 24th, a "scraping

sound" and a "brushing sound;" on December 1st, a "peculiar sound;" and on January 6, 1903, a "crunching sound." On this last day the sound was not so distinct; at times it could not be heard, and the area over which it was heard was smaller than before.

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THE CELLULAR CHANGES IN TUBERCULOUS LEPTOMENINGITIS.*

By I. B. Diamond, M.D.,
Assistant in Medicine, Rush Medical College.

(From the Pathological Laboratory of Rush Medical College, Chicago.)

In view of the interest attached in recent years to the presence of plasma cells and phagocytic endothelial cells and their relation to certain acute infectious inflammations, and since finding similar cells in acute tuberculous leptomeningitis, I have made, at the suggestion of Prof. Hektoen, a histological examination of the tissues at hand in this laboratory, in order to determine, if possible, their rôle in this form of tuberculous inflammation.

The tissues examined are from four cases, three acute and one chronic. Sections, fixed in alcohol and in Zenker's fluid, were stained in Unna's polychrome methylene blue, preceded by eosin, as recommended by Mallory; some were stained in thionin, and others in Harris' carbol-toluidin blue. Clear pictures were obtained with all of these methods.

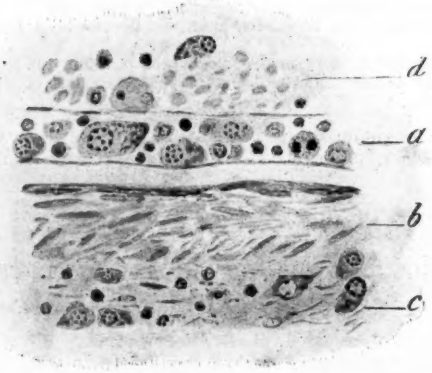
The cellular changes in acute tuberculous leptomeningitis will first

* Presented before the Chicago Pathological Society, May 13, 1901.

be described. These may be summarized as follows: Plasma cells together with lymphoid and phagocytic cells form the greater portion of the cell-infiltrations in the vascular and extravascular areas of the leptomeninx.

The most important of the vascular changes, as shown by Hektoen,[1] is a tuberculous endarteritis, affecting the medium-sized and smaller arteries, characterized by the formation of intimal tubercles and a diffuse, subendothelial intimal proliferation. Examination of such arteries shows the following: The earliest cell change consist in the accumulation underneath the endothelium of the intima of plasma and lymphoid cells, with an occasional polymorphonuclear leucocyte (Fig. 1, *a*). The plasma cells do not differ materially from those

FIG. 1.



a, collection of plasma and lymphoid cells underneath the endothelium of a small artery; *b*, media; *c*, adventitia, containing a few plasma and lymphoid cells; *d*, lumen of vessel. Eosin and polychrom methylene blue. Oil Im. 1/12, oc. iv. (Diamond.)

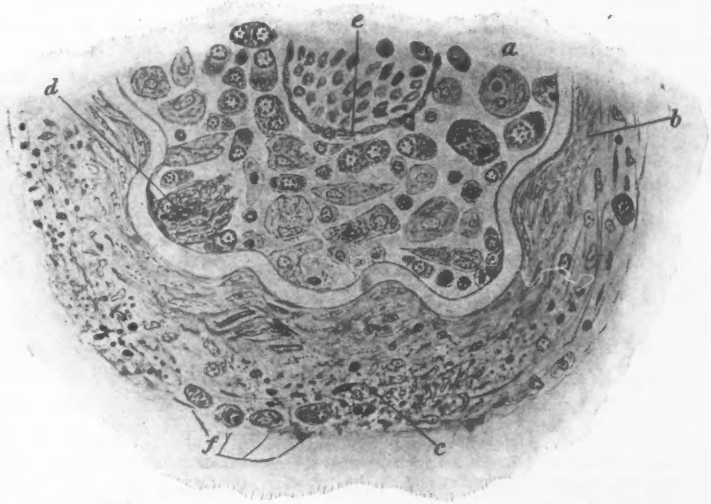
described by Councilman[2] in acute interstitial nephritis. They may be round or oval in shape and vary considerably in size. Nuclear figures are frequently seen (Figs. 1 and 2, *a*). There is usually but one nucleus in a cell, situated eccentrically, but occasionally there are two and rarely three or four in a cell. In the round or young plasma cell the nucleus is usually in the centre. While many of the plasma cells proliferate by mitosis, others, again, seem to be derived from lymphoid cells, as they appear to have the characteristics belonging to both cells.

At first the plasma and lymphoid cells emigrate from the blood or from the lymph spaces of the arterial adventitia, and then find their way underneath the endothelium. On the one hand, passing directly through the endothelial layer, while, on the other, penetrating the

elastic layer. This was especially marked in one of the cases, the tissues of which had been well preserved. Here many plasma and lymphoid cells were seen in the act of passing through the elastic coat from the perivascular lymph spaces into the intima. They were very irregular in shape, many of them elongated, lying, as it were, across the elastic coat, part of their cell body and nucleus being in the intima and part in the media.

In the more advanced stage other cells are found mixed with the plasma and lymphoid cells, namely, the subendothelial *epithelioid* cells. These proliferate from the subendothelial intimal connective

FIG 2.



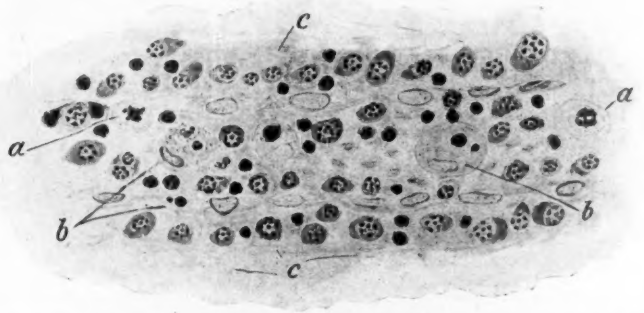
a, collections (underneath the endothelium) of plasma, lymphoid and subendothelial intimal connective tissue cells, also a giant cell at *d*. A few of the cells contain inclusions. Note the karyokinetic figure in the plasma cell; *b*, media; *c*, adventitia, showing caseation; *f*, four shrunken phagocytes; *e*, swollen endothelium. Eosin and polychrom methylene blue. Oil im. 1/12, oc. iv. (Diamond.)

tissue, next to the elastic coat (Fig. 2, *a*). They are large, irregular cells, with granular and vacuolated cytoplasm, and usually eccentrically arranged vesicular nuclei. Mitotic figures are occasionally seen. Under an oil immersion they resemble epithelial cells, and are to a certain degree phagocytic, incorporating plasma and lymphoid cells and occasionally polynuclear leucocytes. These cells may form giant cells (Fig. 2, *d*), and in this manner characteristic intimal tubercles develop.

At this stage the endothelium, which is intact and only lifted up by the collection of these various cells, may now become disarranged and

may also show other changes, especially when there is hyaline degeneration or caseation, either of the proliferated cells or of the outer coats of the vessel wall. The individual cells may be swollen and irregular and their nuclei vesicular (Fig. 2, *e*). Proliferation is rarely, if ever, seen in the endothelium lining the arteries and veins, but may be found to a marked degree in those lining the capillaries and lymph spaces of the pia. Nuclear figures are frequently seen in the endothelium of such capillaries (Fig. 3, *a*). The newly proliferated endothelial cells possess marked phagocytic properties. They are found within the bloodvessels, especially in the capillaries (Fig. 3, *b*) and lymph spaces, in the exudates and throughout the surrounding tissue, where they are very numerous. The cell inclusions consist mostly of lymphoid and plasma cells, and as many as twelve may be present

FIG. 3.



Small capillary: *a*, nuclear figures in the endothelium, the one to the right is free in the lumen of the vessel; *b*, endothelial phagocytes; *c*, collections of plasma and lymphoid cells. Eosin and polychrom methylene blue. Oil Im. 1/12, oc. iv. (Diamond.)

within a single phagocyte. These phagocytic endothelial cells are in every respect similar in appearance with those described by Mallory[3] in typhoid fever.

Plasma and lymphoid cells are also found in considerable numbers in the adventitia of the arteries. In some places they form distinct collections. In the collections, and especially surrounding them, are found many phagocytic endothelial cells and a few leucocytes. Many of the phagocytes contain inclusions of lymphoid and plasma cells. Changes in the intima may or may not be present. Some arteries show caseation of the adventitia with recent cell changes in the intima (Fig. 2). The phagocytic cells which are found in and around such caseated areas are reduced in size, considerably shrunken, and their inclusions are broken up into small fragments (Fig. 2, *f*) while the plasma cells appear as indistinct rings or may resemble epithelioid cells.

In the early stages, when there are no evidences of caseation, the most numerous cells found in the capillaries and lymph spaces, as shown in Fig. 3, are the plasma and lymphoid cells. From here they evidently pass out into the surrounding tissue, where they form the greater part of the cellular infiltration. They are found collected in masses at the sides of the vessel walls, and in some places are so numerous as to cover up entirely the endothelium of the capillaries.

Similar appearances are to be found in the walls of the smaller veins. These are extensively infiltrated with plasma and lymphoid cells. In some areas the collection of cells is so great that the walls are completely covered. When the infiltration is circumscribed there is formed the so-called "lymphoid tubercle," composed, however, of both lymphoid and plasma cells.

Examination of the extravascular areas, the tissue between the blood-vessels and in the interstices of the loosely arranged connective tissue fibres, shows considerable fibrin, in the meshes of which are entangled plasma and lymphoid cells, large phagocytic endothelial cells, and a few leucocytes. Later all these areas may undergo extensive caseation, which even involves the adventitia of the arteries. The latter are then usually thrombosed.

The subjacent cortex is also infiltrated with plasma and lymphoid cells, while the perivascular lymph spaces of the capillaries in the cortex are always distended with plasma cells. Plasma cells are also found in the capillaries of the cortex.

The foregoing is a brief description of the cellular changes found in acute tuberculous leptomeningitis. Of especial interest are the great production of plasma cells and to a less degree of phagocytic cells. So far as I know, plasma cells have never been described in *acute* tuberculous inflammations. It will be seen, however, from the foregoing that the plasma cells play here an important rôle, the cell changes being analogous to a marked degree with those described by Councilman[3] and Pearce[4] as occurring in scarlet fever. Not less interesting is the production of the phagocytic cells. Although less marked it is analogous, on the other hand, to the proliferations described by Mallory[3] as occurring in typhoid fever. In a recent article on proliferation and phagocytosis, Mallory[5] also describes the proliferation of phagocytic cells in the meninges in a case of tuberculous meningitis.

An interesting question is the origin of the phagocytes. Mallory believes that in tuberculosis the proliferated connective tissue cells become phagocytic. This, I believe, is true to a certain extent in tuberculous leptomeningitis. Especially those cells which proliferate from the subendothelial connective tissue of the arteries. It is difficult, however, to determine whether a given phagocyte is of connective

tissue or endothelial origin, for the reason that they cannot be distinguished morphologically, after they become phagocytic.

The process of phagocytosis as first described by Mallory is not yet universally recognized. And various interpretations are given to these cells when they are met with. A case in point is a recent article by Turner[6] on the destruction of nerve cells. Here he illustrates various large cells from the meninges in suppurative meningitis. On page 82 he says: "In cases where recent lymph is found in the meninges, the nuclei segment, and sometimes daughter cells, are seen within the large multinucleated corpuscles which appear to be ultimately extruded, leaving empty spaces in the parent corpuscle." These cells, from the drawings, appear to be nothing else than phagocytes with cell inclusions.

I have since examined, through the kindness of Prof. Hektoen, all his mounted slides of tuberculous meningitis, and have invariably found in these sections phagocytic cells. In some sections they are the most numerous cells present in the extravascular areas. In his article Dr. Hektoen frequently describes them as large cells and as multinucleated giant cells. Plasma cells were also found quite numerous, but difficult to recognize in these preparations. This is especially true with the cells found underneath the endothelium, for they (plasma cells) closely resemble (hæmatoxylin and eosin preparations) the subendothelial intimal connective tissue cells.

Since Mallory[5] describes an instance of the presence of bacteria in phagocytic endothelial cells, I may mention the fact that the large round cells containing tubercle bacilli, and which are described by Dr. Hektoen in the smear preparations from Case V.,[1] are also phagocytes. Many of them contain cell inclusions of lymphoid cells. From this it seems probable that phagocytes must also be reckoned with as a factor in the transmission of tubercle bacilli from one area to another in tuberculous meningitis.

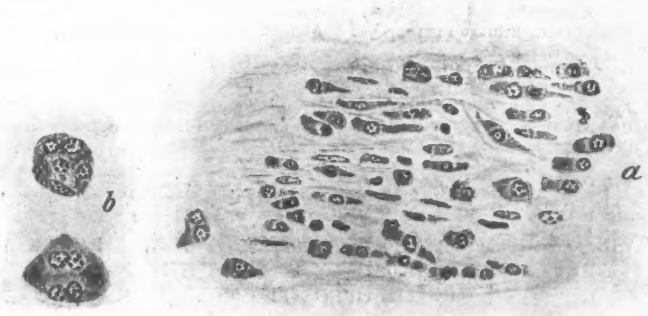
The fate of the plasma cells is another interesting question. In *acute* tuberculous meningitis the majority naturally soon break down and die as a result of caseation. Therefore the tissue from a case of (chronic) healing tuberculous meningitis was studied.* As might be expected, plasma cells were found in great numbers; in fact, they make up the bulk of the proliferated cells. Sections stained with eosin and polychrom methylene blue show, on examination, a pinkish, loosely fibrillated, homogeneous matrix of collagenous tissue in which are embedded mostly plasma cells (Fig. 4). These are mostly elongated and spindle-shaped, and contain usually but one nucleus. Nuclear figures are not seen. Many of the irregular or squarish plasma cells

* This case was described by Dr. Hektoen with reference to the fate of the giant cells in healing tuberculous meningitis. Journal of Experimental Medicine, 1898, vol. iii., No. 1.

contain two to four nuclei. Giant cells are frequently seen, containing from four to eight nuclei (Fig. 4, *b*). These evidently multiply by indirect division, as many of the cells contain constricted or kidney-shaped nuclei, similar in appearance to those described by Krompecher.[7]

A certain number of the plasma cells show various degenerative changes, which are similar to those described by Schottländer and Krompecher.[7] These are the transformation of plasma cells into epithelioid cells, hyaline degeneration—round homogeneous violet-stained bodies within the plasma cells—and vacuolations. The majority, however, are undergoing progressive change, namely, changing into connective tissue cells. This, I believe, is evident from the character of the tissue—granulation tissue—and from the appearances of the plasma cells themselves. Transition stages between the two cells are

FIG. 4.



a, plasma cells resembling connective tissue cells; *b*, giant plasma cells. From (chronic) healing tuberculous meningitis. Eosin and polychrom methylene blue. Oil im. 1/12, oc. iv. (Diamond.)

frequently seen. As the cells elongate and become spindle-shaped the perinuclear spaces in the plasma cells become larger, while the granular cytoplasm is heaped up in clumps peripherally in the cells. Harris[8] interprets these appearances of the plasma cells to a secretion of fibrous tissue instead of a direct transformation of the cells into connective tissue.

A very interesting question is: Are all the plasma cells in this tissue derived from lymphoid cells and mononuclear leucocytes, or are part of them of connective tissue origin? as Joanovicas[9] claims, and that only those derived from connective tissue cells are able to form new connective tissue. This question must remain unanswered. It will be seen, however, that the plasma cells in acute tuberculous inflammation differ somewhat from those present in the chronic form.

Although conclusively shown by most observers that plasma cells are derived from lymphocytes, opinions still vary. Recently Herbert[10] makes the statement that lymphocytes are young plasma cells—daughter plasma cells—while the plasma cells are derivatives of the fixed connective tissue cells. Comment, however, is unnecessary.

In conclusion, I wish to express my thanks to Prof. Hektoen for kind advice during the course of this investigation and also for the use of his microscopic slides.

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A CASE OF PARALYSIS OF THE SIXTH AND SEVENTH CRANIAL NERVES OF THE RIGHT SIDE WITH HEMIPLEGIA OF THE OPPOSITE SIDE.

By E. Andrade, M.D.,
ex-assistant to the ophthalmic clinic of the university of genoa (italy).

The study of the paralysis of the motor nerves of the eye is very important, not only on account of the alarm which they produce in the patient, the æsthetic effect and the visual disturbances, but especially by reason of their often being the first symptoms of serious central disorders. Since the ocular nerves have circumscribed nuclear origin and a well-known course, the alterations of their function very often allow the definite localization of the lesion. If at the same time other paralyses are present the point where the pathological process is located can be determined even more accurately.

One of the most frequent paralyses of the motor nerves of the eye is that of the third nerve, its presence being a very important sign in many nervous diseases. In certain cerebral disorders, paralysis of the oculomotor, especially of those branches that supply the exterior muscles of the eyeball, is accompanied by hemiplegia of the opposite side, and this complex symptom called Weber's syndrome, is a positive indication of a

lesion of the inferior part of the cerebral peduncles, or of the neighboring portion of the pons, on the same side of the ocular paralysis. The knowledge of the course followed by the oculomotor from its origin in the several cellular groupings on the floor of the aqueduct of Sylvius to its exit on the inner side of the cerebral peduncles, near the apex of the interpeduncular space, will easily explain this localization.

But besides Weber's syndrome there are other crossed paralyses which permit the locating of pathological processes at different points of the pons, as will be seen in the following case which I have had occasion to study during some time:

Clinical History. M. B., colored girl, aged ten years. Father contracted syphilis two years before her birth; mother has had six miscarriages, two before and two after the birth of the child. The girl was born with the skin of the hands and feet macerated, the mother saying that they looked as if she had on white gloves and stockings, but otherwise she seemed to be in good health until two years ago, when symptoms made their appearance which compelled her to consult an oculist.

It was then observed that her right eye was strongly turned in—internal squint. The oculist who saw her performed two operations on her eyes (tenotomy and advancement?) without any good result. A few days after the last operation, which took place in November, 1901, it could be plainly seen that the left arm and leg were weak, having lost most of the strength necessary to perform the usual movements, and that the left labial commissure was drawn up when the child smiled.

In the last three months the patient has been afflicted with attacks of vomiting and vertigo three or four times, the vertigo being so strong as to cause her to fall down. Headache, s metimes of a violent character, is a constant and annoying symptom. The child has progressively lost her vivacity and is now in a noticeable condition of mental depression.

The eye fundi were normal at a first examination, but twenty days later there was congestion of the disks with dilatation of the venous vessels and prominence of the papillæ, all more marked in the right eye. The pupillary diameters and reactions show no deviation from normal.

Studying the movements of the eyeball, it is seen that the right external rectus muscle is completely paralyzed. When the eyes are directed toward the right side, the centre of the right cornea does not pass beyond the middle point of the inferior lid. All the other muscles work regularly, but in extreme lateral positions of both eyes a marked horizontal nystagmus is present. There are no associated paralyses of the movements of the eyes (Blicklähmungen). There is homonymous diplopia, especially when the eyes are directed toward objects situated on the right side of the patient. Olfaction, taste, and hearing are normal. Pharyngeal sensibility is decreased somewhat.

The left arm and leg are paralyzed, but they can with difficulty perform some movements. There is no anæsthesia or analgesia and thermal sensibility is not altered. There are no contractions. The knee-jerk is enfeebled; all other reflexes are normal.

The right facial nerve is paralyzed, but only in its inferior portion, the frontalis and orbicularis muscles showing no functional disturbance. No deviation of the tongue can be detected, which proves that the

fibres of the hypoglossus are not affected. Though the child answers intelligently all questions put to her, there is a condition of mental apathy.

The foregoing symptoms, I believe, are caused by a syphilitic gumma situated in the inferior region of the pons on the right side.

The complex symptom just described in the preceding clinical history is characterized by the paralysis of the external rectus and facial nerves on one side and hemiplegia of the opposite side. In some other cases that have been reported there was besides paralysis of the hypoglossus and of the motor portion of the trigeminus; and sometimes, instead of hemiplegia, there has been tremor or hemianæsthesia. All these cases are forms of what is called Millard-Gubler's syndrome, which consists essentially in facial paralysis, more or less complete, with hemiplegia of the opposite side (crossed paralysis), and which is considered one of the most constant manifestations of disease of the pons.

In order to understand the reasons which have prompted me to localize the pathological process in the inferior region of the pons, on the same side of the ocular paralysis, it is necessary to remember the origin and course of the nerves which are supposed to be involved in the lesion.

The sixth nerve originates in a nucleus situated in the floor of the fourth ventricle surrounded by the radicular fibres of the facial, the nucleus of which is located more deeply in the floor of the same ventricle. From its origin the fibres of the abducens united in three or four bundles take a dorsoventral course parallel to the median raphé. They continue through the trapezium and peduncular fibres to appear as a single trunk at the base of the brain in the groove between the anterior pyramid and the inferior border of the pons. The radicular fibres of the facial nerves after leaving their nucleus ascend loosely connected toward the median line, forming the ascending branch of the root of the facial, and turning forward they cover the dorsolateral aspect of the abducens nucleus. At this point the facial reaches very near the floor of the ventricle, where it produces an elevation—tuberculum nervi facialis.

The facial fibres are then directed downward, passing before the anterior end of the nucleus of the sixth nerve (descending branch of the root of the facial), and farther down between their own nucleus of origin and the sensitive root of the trigeminus. They make their exit at the base of the brain in the lateral part of the groove between the medulla and the pons.

Though the relation between the nuclei and radicular fibres of the sixth and seventh nerves are very intimate, recent investigations have failed to support Meynert's contention, which states that many fibres of the facial, those which supply the frontalis and orbicularis muscles, have their origin in the abducens nucleus. In fact, it has been demonstrated

that in cases of complete rupture of the facial nerve in the aqueductus Fallopii, none of the cells of the nucleus of the sixth nerve have shown any secondary degeneration. According to the majority of the investigators, however, the abducens nucleus sends to the oculomotor of the opposite side fibres which supply the internal rectus in the lateral associated movements of the eyes.

This anatomical description explains without any difficulty why a lesion of the pons at its inferior region, where the fibres of the sixth and seventh nerves come into relation with the peduncular fibres that cross lower down at the decussation of the pyramids to supply the opposite side of the body, would cause all the symptoms met in the case just reported. I do not believe that the pathological process affects the nuclei, because there is no paralysis of the associated movements of the internal rectus of the opposite side, as would be the case if the nucleus of the sixth nerve were diseased.

The limitation of the facial paralysis to the inferior portion of the territory supplied by the seventh nerve without involvement of the frontalis and orbicularis muscles does not necessarily indicate a nuclear lesion, because the fibres of the facial while going through the pons are very loosely connected and it may easily happen that a lesion destroys only those which supply the inferior territory.

In regard to the nature of the lesion, the family history makes it very likely that we have to deal with a syphilitic gumma.

Hereditary syphilis, it is well known, can produce this pathological change in the nervous system. In spite of certain symptomatic differences, more apparent than real, between hereditary and acquired syphilis, the pathological process is the same, an accumulation of cells that causes arteritis, thickening of the nerve sheaths, or a gumma.

The slow and progressive course of the disease compels us to discard the idea of a hemorrhage due to syphilitic endarteritis. The increased congestion of the optic disks during the preceding week points to an extension of the granuloma. Notwithstanding all that has been said in favor of a syphilitic lesion, it is not wise to exclude entirely the possibility of a tubercle of the meninges of the base of the brain, that, pressing on the pons in the region of the sixth and seventh nerves, and destroying the corresponding fibres, would produce all the symptoms just described. Hereditary syphilis does not exclude tuberculous meningitis. In this case, however, the paralysis of the facial nerve would be complete, affecting all the muscles supplied by it, including the frontalis and orbicularis. The result of the energetic antisyphilitic treatment to which the patient has been submitted will help, no doubt, to clear the etiology of the disease, but we must remember that cerebral syphilis in an advanced stage does not always respond to antisyphilitic measures.

HICCOUGH. REPORT OF A CASE.

By L. W. Atlee, M.D.,
OF PHILADELPHIA.

A COMPLETE study of hiccough from a physiological and pathological point of view remains yet to be made. From a clinical standpoint this is much more worked out. Historically the subject is one of great interest, as it has been observed and commented on among our earliest medical writings; in fact, the bibliography is very voluminous, and though of use to those whose tastes lead them into such investigations, they will reward us with little of practical value, as the observers were more led by theoretical ideas than by observation of facts—a reproach, indeed, from which modern medicine cannot by any means escape.

R. M., American, aged seventy-nine years, a large, well-nourished old man. One week ago he began to hiccough; he could attribute no cause for it. When first observed during the earlier three days there would be intervals when the condition ceased entirely, but for the past four days he complains that he has hiccoughed uninterruptedly night and day. At first he had lived on his ordinary meals and diet, but later this had caused vomiting in addition to the already existing hiccough, and for the past couple of days he had taken nothing but tea and crackers. When first seen—on the eighth day—his countenance bore a very anxious expression. The color of the skin and mucosa suggested cyanosis from defective aëration of the blood due to the inefficient respiratory efforts. The tongue was coated with a thick, white, pasty fur; his bowels had not moved for the past four days. Examination of the thoracic organs revealed nothing abnormal beyond some emphysema, so common in stout, aged people. The heart was large, but no murmurs could be detected. Examination of the abdomen showed nothing excepting double inguinal hernia. These were large and descended well into the scrotum. They were easily reduced and retained by compresses and a spica bandage of the groin. In fact, this was done immediately on their discovery, as such a condition was very suggestive as a causative factor of the vomiting, hiccough, and constipation, though there was no abdominal distention; but the replacement of the herniæ produced no amelioration of the symptoms.

The medical treatment was begun by the time-honored giving of a large calomel and soda purge. A mustard plaster was applied to the epigastric and left hypochondriac regions (a very large one), and kept on as long as it could be borne. After the calomel had acted well, he was given a mixture containing bismuth subnitrate, dilute nitric acid, and spirits of chloroform in compound tincture of cardamom; this was to be given every hour while the hiccough lasted, and to be repeated should it return. The diet, which had hitherto been restricted in no way, was to be limited to the white of an egg stirred up in six ounces of cool water, alternating this every other feeding with clam-broth or juice. (The best and simplest manner of obtaining this is by heating six large, well-cleaned clams, unopened, in a pot; as soon as they open

the juice is strained off; if too salt, it can be diluted with a few tablespoonfuls of water.)

After the fourth dose of the mixture and pretty severe counterirritation by the mustard plaster, the hiccough ceased, and did not recur for three hours, when it was again controlled by the mixture, so that he passed a whole night without any hiccough. The following morning, unfortunately, it began again at an early hour more severely than ever. A repetition of the former treatment being unsuccessful, he was given twenty grains of chloral by the mouth. In the course of ten minutes it began to exert its well-known power to stop such spasmodic movements, and in half an hour the hiccough had entirely ceased. This interval lasted eight hours, when a second dose of the same size was given, almost immediately controlling the spasmodic movements. Subsequently the chloral had to be repeated twice, when the tendency to a recurrence of the hiccough ceased.

It is now some three months since this case was under observation, and the old man is enjoying his usual extraordinary good health.

In old or enfeebled persons hiccough can be the direct cause of death, which is attributable to the effects of the long continuance of the diaphragmatic choreic spasms on the performance of vital functions. This is markedly seen in its action on respiration, causing deficient aëration of the blood. As the disease is usually aggravated by the ingestion of food, the patient will soon begin to suffer from the deprivation of needed sustenance. Again, it is impossible to sleep owing to the violent sudden commotions given to the whole body at each spasmodic descent of the diaphragm.

The acts which take place and occur in succession to produce the phenomenon of hiccough are contraction of the diaphragm, convulsive agitation of the abdomen and thorax, vibration of the glottis producing a characteristic sound. The contraction of the diaphragm is the initial act; it is brisk, spasmodic, and involuntary, and consists in tension of its mass and its descent, and has for its effect the crowding of the contents of the upper abdomen. The respiratory movement determines a rush of air through the respiratory tract, which is sudden and violent. This column of air coming in contact with a flaccid glottic orifice produces the accompanying sound, as the normal glottic dilatation of inspiration does not take place. These phenomena repeat themselves many times.

All the muscular phenomena which go to make up the respiratory act are innervated in the centre for this movement in the medulla, but being as it is a most complex act, there are a great variety of central, intermediary, and peripheral places where stimuli may produce disorder of the proper concensus of the muscular actions.

Direct irritating lesions of the respiratory centre, either inflammatory or from neoplasms, have been accompanied by hiccough as a symptom. The hiccough seen during the infectious diseases, or in

defective elimination of physiological products, can be viewed as of central origin, due to the effect of poisons circulating in the central nervous system. As an example of hiccough from the effect of toxic agents, may be mentioned the *hoquet tobagique*, of the French, due to tobacco poisoning.

Any irritation of the phrenic nerve from its origin in the cervical plexus, or of its fibres in the cervical cord, may cause this phenomenon, as it has been observed in transverse myelitis of this region, associated with slow pulse, dysphagia, dyspnoea, and myosis. It has also been observed during the course of neuralgia of the branches of this plexus (probably a neuritis). Direct irritation of the phrenic nerve by tumors of the mediastinum, aneurisms, pneumonia, and pleuritic effusions (if the mediastinal pleura is involved), may cause hiccough. The same may be said of the nerve in its course before entering the mediastinum, where it may be irritated by enlarged glands, abscess, tumors, or aneurisms.[1]

The frequent occurrence of hiccough during the course of diaphragmatic pleurisy has led Geuneau de Mussy to regard it as being caused by a neuritis of the terminal filaments of the phrenic nerve. Though it has not been observed as an unfailing accompaniment of this disease, it has been seen frequently enough to make it suggestive of this condition.

Since hiccough is (in by far the greatest number of cases) caused by abdominal derangements, we must agree with Gowers[2] "that the curious phenomena of hiccough may be connected with affections of this nerve (pneumogastric), as they certainly are with the respiratory centre." Perhaps the most common cause of hiccough is an overfull stomach; and next most frequent, distention of the stomach with "wind," or, as Hippocrates puts it, "*Convulsio fit aut a repletione, aut ab evacuone, sic etiam singultus.*" In many people the ingestion on an empty stomach of hot drink or food will bring on a paroxysm, as will also brandy, liqueurs, or food highly seasoned with red pepper, and such like condiments. It is met with in all forms of stomach disease accompanied by digestive difficulties, catarrhal, ulcer, cancer, and gastralgia. "The existence of hiccough is a sign of some value in cancer of the stomach when the question arises in cases where the growth cannot be palpated, as it is a not infrequent symptom of cancer of the cardiac region or vicinity."[3]

All forms of irritation or inflammation of the intestinal mucosa are frequently attended with hiccough, indigestion, worms, dysentery, etc.

[1] Rosenthal. Diseases of the Nervous System, vol. ii. p. 225.
[2] Diseases of the Nervous System, vol. ii. p. 295.
[3] Dict. Ency. des Sci. Med., T. xiv. p. 425.

Irritation and inflammation of the liver and its gall-bladder or ducts are also among the frequently observed causes of hiccough. Any cause of obstruction to the onward movement of the intestinal contents is usually accompanied by this affection, as in paralysis of the intestinal walls, or in obstruction of the continuity of the intestinal tube by its contents, or from causes outside the tube in the form of bands, neoplasms, etc., or by volvulus, intussusception, strangulated hernia, etc. It is common during the course of acute peritonitis, and from observation of it in this disease and during the course of intestinal obstruction among its terminal manifestations, Hippocrates regarded it as a sign of evil omen—"*ex vomitione singultus, Malum.*"

Hiccough occurring during the course of typhoid fever or any ulcerative intestinal disease should suggest perforation of the intestine.[1] The disease is frequently observed in uræmic states, and very often is among the terminal symptoms in genito-urinary infections, as in the pyelonephritis arising from bladder infection in chronic cystitis due to prostatic obstruction. In the first, we are dealing with a hiccough of central origin due to the toxæmic condition; in the latter, both peripheral and central causes can be factors in the hiccough.

Among the protean manifestations of that capricious neurosis—hysteria—hiccough is a not infrequent symptom, and may be regarded as of central origin. In this class may be placed those cases where the hiccough has followed fright or some violent emotion. Among the older writers on this subject instances are recorded where the hiccough was of several years' duration, a fatal result being averted by occasional cessations of the spasmodic movements. The application of the well-known physiological law of the tendency to repetition of accustomed movements will be suggested by the long continuance in these cases.

Owing to the great variety of conditions which may give rise to hiccough, the means found successful in its treatment has been of the most extreme diversity; but naturally its treatment consists in the removal (if possible) of the morbid condition giving rise to it. At the same time we may have to treat it entirely symptomatically, for, as we have previously stated, it may be productive of a condition in debilitated persons resulting in death.

In the simplest form, as seen, for instance, in infants, and due to overdistention of the stomach with food or wind, emesis or a carminative is indicated.

Occuring in adults and due to simple gastric irritation, large doses of bismuth subnitrate, perhaps combined with some aromatic substance, as the compound tincture of cardamom, and spirits of chloroform, will

[1] Quain's Dictionary of Medicine, article Hiccough.

be often successful. Small doses of dilute nitric acid added to these are seemingly useful also.

When a case proves rebellious to remedies directed only to the correction of the apparent cause of the hiccough, we may then resort to medicines of a purely antispasmodic character. Of these chloral has been found to be pre-eminently the most effective, and should be given in a full dose. As much as twenty grains can be repeated at hourly intervals until sixty grains have been taken. Should the hiccough still continue, it is probable the chloral will not be successful in larger and also, perhaps, dangerous doses. The bromides have not shown themselves so useful in the cases where gastric irritation was largely the factor in its production. In these cases it is of the greatest benefit to apply counterirritation to the epigastrium, for which a mustard plaster is usually the most readily found to hand. The diet in these cases is also of the first importance, and must be of the blandest description: albumin water, weak chicken tea, or clam juice or broth, until the gastric irritation has been allayed.

It would be to no purpose to enumerate the great variety of drugs that have been advised for hiccough. Among the many means recommended, however, several are worthy of mention as having been frequently successful. It has been known that the act of sneezing will break up the morbid train of phenomena that go to make up the act of hiccoughing. To provoke it, remedies called sternutatories were used. The easiest one of these to find is ordinary tobacco in the form of snuff.

As any violent emotion can produce hiccough, so it has also been known to stop it. Dupuytren cured a protracted case instantly by the application of the actual cautery to the epigastrium—a remedy, we doubt, would be submitted to by many.

Of mechanical measures, direct pressure on the phrenic nerves at the root of the neck, forced flexure of the head on the thorax, and deep, continuous pressure in the epigastrium are said to have caused a cessation of the spasm.

When hiccough arises in hysterical subjects, it is well to begin with the milder antispasmodics, such as valerian or the valerianates, passing on in case of non-success to more powerful drugs—chloral, apomorphia, or hyoscine hydrobromate. Counterirritation of the upper cervical spine, or of the epigastrium, ovarian pressure, and spraying the epigastrium with ether have all been successful, but usually fail when frequently repeated.

The hiccough due to sudden large losses of fluid from the body by hemorrhage, or in Asiatic cholera, is relieved by its restitution through the means of normal saline solution introduced in any of the various ways—entero, hypodermo, or venous.

CLINICAL GONORRHŒA WITHOUT THE GONOCOCCUS.

By A. L. Benedict, A.M., M.D.,
of Buffalo.

While the existence of urethritis, colpitis, and balanoposthitis not due to the gonococcus has been commonly admitted for a number of years, my attention has been directed to the matter by the paucity of actually reported cases. In a fairly systematic review of the world's literature for the past four years, I have thus far found only one note on the subject, namely, an article by Malherbe on "Staphylococcus Urethritis," in *Annales des Maladies des Organes Genito-urinaires*, November, 1901, abstracted in The American Journal of the Medical Sciences, March, 1902. Unquestionably, however, other articles must have appeared on this subject.

In several instances in which, for medico-legal, moral, or therapeutic purposes, discharges from little children have been submitted to me for examination, gonococci have been found without explanation of their source, and in apparently identical cases they have been absent. Of these cases I have no exact memoranda and the patients were not under my direct observation:

H. A. B., seen in 1889, presented the ordinary picture of acute gonorrhœa, with free discharge. He denied having had gonorrhœa in the past or of having had extramarital intercourse. His wife had some leucorrhœa, according to his statement.

An unknown patient, 1901, presented a swollen and œdematous prepuce, phimotic, and with a sanious discharge from between the prepuce and the glans. The same discharge could be squeezed from the urethra in small quantities, but appeared merely to have entered from without. He denied previous gonorrhœa or recent possibility of infection, except a single intercourse with his fiancé, whom he had every reason to suppose a virgin until that time. He was a man of refinement and was, naturally, much distressed.

Miss ——, 1902, complained of acute cystitis, with pre-existing leucorrhœa, for which she had used a clean syringe; the vesical symptoms followed in a few days. The urethra was tender to the touch, and the urine deposited a bulky, purulent mass. She claimed to be a virgin, although the hymen was not palpable and the vagina was capacious. For extramedical reasons, ocular examination, catheterization, and definite localization of the source of discharge were not instituted. The urine, however, after a vaginal douche, still contained an abundance of pus, and the clinical evidence of the joint involvement of the bladder and urethra is ample.

None of these cases presented evidence of the presence of the gonococcus, according to the ordinary Gram and decolorization tests. Indeed, by the simplest staining methods there was nothing to suggest the gonococcus, but the tests were, nevertheless, made. Various and numerous cocci, singly and in chains, and short and long, thread-like bacilli were present in all cases, but were not identified. All three cases yielded to treatment, consisting, respectively, of the ordinary syringing, local application of hydrogen peroxide, and use of urinary antiseptics. The first case was rather refractory.

In this connection may be reported the case of an intelligent Pole, who presented a peculiar, indurated and, in places, glossy glans, but no urethritis. Although married, he had not had intercourse for six months, and he denied venereal taint. His condition proved to be a mycosis, the fungus consisting of a mass of mycelia, without spore-bearers. Neither myself nor Dr. Herbert Upham Williams could identify it exactly.

No apology is called for in regard to the first case, as I was then in general practice. The second patient was from out of town and was kept waiting until after ordinary office hours through a misunderstanding of his condition, which he referred, vaguely, to his abdomen. As his time was valuable, I undertook to make the preliminary examination and to refer him, if his disease proved to be gonorrhœa. The third case was an intercurrent affection in an intestinal toxæmia, with constipation and putrefaction in the bowel. While the syringe seems to have been the immediate carrier of infection, the case may ultimately have been due to the colon bacillus. The fourth patient was kindly referred to me by Dr. Lucien Howe, and suffered from gastric dilatation and catarrh. The scraping from the glans was examined as a matter of interest.

REVIEWS.

A System of Clinical Medicine, Etc. By Thomas D. Savill. London: J. & A. Churchill, 1903.

The plan of this work is quite original and deserves a detailed description. The Gordian knot of scientific classification is boldly cut by "considering convenience and practical utility rather than an appearance of scientific precision." Not that traditional lines have been altogether abandoned; in the main the classification—the infectious fevers excepted—is by physiological systems, but the starting-point in the description of each disease is always a prominent group of symptoms common to a number of different pathological conditions, and not the title of any special disease. After all the possible causes that might be responsible for the particular symptom group have been passed in review from the purely diagnostic standpoint, each disease is described systematically under the heads of diagnosis, prognosis, and treatment, with an occasional reference to the pathology for purposes of elucidation. Theoretic discussion is avoided as much as possible. For example, after a short description of the general symptomatology of diseases of the abdomen and the methods of physical examination employed in that region, we find the diseases of the abdomen collected as "causes of abdominal pain" into two groups, the one entitled "Abdominal pain coming on suddenly with collapse," the other "Abdominal pain coming on suddenly without collapse," in both of which appendicitis figures. After this general division of the subject a series of special symptom-groups are detailed as: "The patient complains of acute abdominal pain which has come on suddenly, with symptoms of severe collapse, attended by vomiting and constipation. The pulse is rapid (over 100). The case is probably one of three conditions: perforation into the peritoneum, acute peritonitis, or acute intestinal obstruction." And, finally, these three conditions are described in regular order, the introductory paragraph being slightly varied or amplified for each disease. This method obviously imitates the actual process of reasoning employed at the bedside and in office practice as closely as it is possible to reproduce a practical procedure in print, and has the great advantage of preserving the relative value of diseases, which is often lost sight of in systematic text-books. After the obvious and relatively frequent conditions have been fully treated, the rarer causes of the clinical picture under consideration are enumerated and briefly described, their lesser importance being reflected in the selection of smaller type.

It is true that the arrangement involves some repetition, for which the author apologizes in the introduction, and makes it somewhat more difficult to turn to the section treating of a given disease; but, on the other hand, the practical problems in diagnosis are presented as they are actually met in practice, and the student is taught to reason from effect to cause. It is an excellent way to enforce habits of observation and logical deduction, and to guard against the vice of establishing a pre-

conceived theory in making a diagnosis. The subject of treatment occupies a subordinate place in the scheme of the book, without, however, being altogether neglected; but the chief merit lies in the part devoted to diagnosis.

The book is distinctly the author's own product. There is not much space devoted to historical details or the theories of his predecessors and contemporaries; it is, in fact, the result of his own observation and experience. This personal quality is most attractive and gives the work a stamp of originality at once distinguishing it from the usual run of text-books. One may not accept everything that the author says, and his peculiar method may not appeal to everyone, but some new light, at least, will be obtained on any perplexing problem for a solution of which the book is consulted. R. M. G.

CANCER OF THE UTERUS. A Clinical Monograph on its Diagnosis and Treatment. With the After-results in Seventy-three Cases Treated by Radical Operation. By ARTHUR H. N. LEWERS, M.D. Lond.; F.R.C.P. Lond.; Obstetric Physician to the London Hospital, etc. With 51 original illustrations and 3 colored plates. 8vo., pp. 321. Philadelphia: P. Blakiston's Son & Co., 1902.

THIS excellent monograph is of great practical value from a clinical point of view, because it is an analysis of the writer's large experience in the treatment of uterine cancer. The illustrations are excellent, and the conclusions arrived at are in accord with the generally accepted views of the best authorities. The diagnoses in all of the cases operated upon were confirmed by the microscope, and the results obtained were interesting and suggestive. There were 33 high amputations of the cervix, with 18 per cent. cured; 28 vaginal hysterectomies for cancer of the cervix, with 14 per cent. cured, and 11 vaginal hysterectomies for carcinoma of the fundus, with 45 per cent. cured. Abdominal pan-hysterectomy is admitted to be the operation which offers the best chances of permanent cure. The writer pleads strongly for the early recognition of cancer, and dwells upon the importance and significance of the early symptoms. A careful study of this valuable contribution to the subject of cancer of the uterus will repay all who are interested in the matter. J. B. S.

THERAPEUTICS OF INFANCY AND CHILDHOOD. By A. JACOBI, M.D., LL.D. Third edition. Philadelphia and London: J. B. Lippincott Co., 1903.

THE third edition of Dr. Jacobi's well-known treatise shows evidence of careful revision and many additions made necessary to bring the matter up to date, but no actual changes in the general character of the book have been made. Radical changes, indeed, were not to be expected. The first edition presented the embodiment of the mature

judgment and unrivalled clinical observation of nearly a half century of active professional life, and in the two subsequent editions little need has been found to alter the opinions based upon so solid a foundation. In no portion of the volume is this fact more suggestively exemplified than in the portion devoted to infant feeding, in which his earlier teachings receive gratifying confirmation at this later date from some of his erstwhile most prominent critics and opponents.

The general scope and arrangement of the work are already too well known to need extended mention. Every page bears the stamp of Dr. Jacobi's individuality, which is often tinged with a degree of dogmatism that would sound rather strange from anyone else. This, indeed, is one of the charms of the book, for it gives to its teachings a touch of finality that at once inspires confidence and challenges respect.

As a contribution to the practical therapeutics of infancy and childhood, Dr. Jacobi's work as it now stands will long continue to occupy a unique and prominent place in pediatric literature. T. S. W.

THE AMERICAN TEXT-BOOK OF OBSTETRICS. FOR PRACTITIONERS AND STUDENTS. By James C. Cameron, M.D.; Edward P. Davis, M.D.; Robert L. Dickinson, M.D.; Henry J. Garrigues, M.D.; Barton Cooke Hirst, M.D.; Charles Jewett, M.D.; Howard A. Kelly, M.D.; Richard C. Norris, M.D.; Chauncey D. Palmer, M.D.; George A. Piersol, M.D.; Edward Reynolds, M.D.; Henry Schwarz, M.D.; J. Clarence Webster, M.D. RICHARD C. NORRIS, M.D., Editor, and ROBERT L. DICKINSON, M.D., Art Editor. With nearly 900 illustrations. Second edition, revised. In two volumes. Philadelphia and London: W. B. Saunders & Co., 1902.

THIS splendid work is so well known that it needs no introduction. For convenience of handling it appears in two volumes, and in this edition there are evidences of careful revision. As an example of bookmaking, printing, illustrating, and editing, and from the standpoint of clear and concise teaching the book is unsurpassed by any work of the kind with which we are familiar. J. B. S.

MUCOMEMBRANOUS ENTEROCOLITIS. By MAURICE DE LANGENHAGEN. London: J. & A. Churchill, 1903.

IN this miniature volume, attractively bound in red, which may be read during a day's round of professional calls, the author gives his readers the benefit of an unusually concentrated experience of this not very common disease among the guests of a popular health resort which is his field of activity. Mucomembranous colitis is not a rare disease; or, at least, like many other conditions, it is now more frequently recognized, and thus appears to be on the increase; the author pleads for its more general recognition in ordinary medical manuals. Outside of France and Germany the disease has been studied a good deal by

American writers; indeed, the name "membranous enteritis" is a contribution from Da Costa. The subject is treated systematically in ten chapters, and the author apparently leaves little to be added. The question of treatment, which is very fully discussed in the last chapter, is an important one in this disease, which "can always be considerably relieved, and sometimes completely cured." It is, moreover, the part of the subject that appeals most to the general reader. There are three indications—to regulate the diet, provide for a daily evacuation of the bowels, and to relieve the pain and concomitant dyspeptic symptoms. The keynote in the choice of foods is the avoidance of fats and any articles that leave a large quantity of ash to irritate the bowel. Green vegetables, salads, and raw fruit are rigidly excluded from the diet list, as the author believes they are not only useless as laxatives, but absolutely harmful on account of their irritative properties. There should be no hesitation about the employment of medicinal laxatives, even daily, if necessary. The author himself prefers castor oil, but the patient's individual peculiarities must be consulted. The local treatment, consisting of high enteroclysis, is an essential feature, and the technique and pharmacology are fully given. R. M. G.

ANATOMY. A MANUAL FOR STUDENTS AND PRACTITIONERS. By WILLIAM H. ROCKWELL, Jr., M.D., formerly Assistant Demonstrator of Anatomy in the College of Physicians and Surgeons, Columbia University, New York. Series of Pocket Text-books, edited by BERN B. GALLAUDET, M.D., Demonstrator of Anatomy and Instructor in Surgery, College of Physicians and Surgeons, Columbia University, New York; Visiting Surgeon, Bellevue Hospital, New York Illustrated with 70 engravings. Philadelphia and New York: Lea Brothers & Co.

THE author states in his preface that the object in writing this volume is to provide the student with a manual at once compact and fairly complete, and this, after a careful review of the book, it must be granted the author has accomplished. *Gray's Anatomy* has been followed as a standard, but it will be noticed in examining the work that repetition, which is so frequently met with in *Gray*, has been carefully avoided. It is a decided advantage to many who will use this book that *Gray's Anatomy* has been followed, because this is a work which has always been familiar to the greater number of men who are practising medicine at the present time, and who, in addition to students who are preparing for examination, will take advantage of this manual to refresh their memories upon important points. It will be noticed that the headings of the subjects are more uniform in this work than formerly, so that, in addition to osteology, arthrology, and myology, we have angiology, neurology, and splanchnology. The most important feature of an anatomy is accuracy of description, and, after carefully examining this work, the writer is unable to detect any anatomical errors. It is to be recommended to both students and practitioners. J. K. Y.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

PROFESSOR OF MEDICINE IN THE JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND,

AND

W. S. THAYER, M.D.,

ASSOCIATE PROFESSOR OF MEDICINE IN JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND.

The Cause and Specific Treatment of Hay Fever.—DUNBAR (*Deutsche med. Wochenschrift*, 1903, xxix. p. 149) contributes a most important article upon the results of his studies, which have been carried out during the last seven years, upon the nature and treatment of hay fever. These are to be set forth more completely in a work which will shortly be published.

Recognizing the fact that hay fever depended largely upon individual predisposition, a great proportion of the people being entirely insusceptible to it, great variances of opinion have existed in the medical profession with regard to the immediate causative agent. Summer heat, dust, or other purely mechanical substances, emanations from grass or hay, and finally micro-organisms have been incriminated. The researches of Elliotson and Blackley suggested that hay fever was caused by the pollen of plants. Thirty years ago this was the more generally recognized idea. Recently, however, the theory that it was due to an infectious agent has been more popular. The author recognized the fact that the question could never be settled until the causal agent could be obtained in pure form, until the disease could be produced at all times of the year in susceptible individuals, and, further, until it was proven that this causal agent was efficacious only in individuals who were known to be susceptible to hay fever. In this task he has apparently met with complete success. Dunbar has shown that the pollen granules of rye, oats, wheat, rice, corn, and all other forms of grass which he has examined contain substances which bring on all the appearances of hay fever in individuals susceptible to the disease, but are entirely without effect upon other individuals. The pollen granules of all other forms of plants—for instance, those of linden, rose, vermuth, and many other plants which have been supposed to be dangerous, have proved to be entirely innocuous.

It was further shown that these pollen granules produced results similar to those generally recognized in the mucous membrane supplied by the

N. trigeminus, when applied to any other part of the body—for instance, to the mucous membrane of the rectum. The same effects were observed at all times of the year. The active substance in the pollen granules was not to be extracted by alcohol or ether, but was more or less soluble in water or physiological salt solution, very soluble in tears, saliva, nasal secretion, and blood serum.

The author believes that the active substance is represented by the starch rodlets, which form the main part of the contents of the pollen granules of grass. These starch rodlets do not apparently consist of pure starch; the active substance would appear to be an albuminoid body. Numerous experiments made by the author and by Thost and Schultz in Heligoland have proven that the application of pollen in pure form or in the shape of solutions to mucous membranes produces without exception the characteristic symptoms of hay fever at all times of the year in individuals susceptible to the disease, while such application is absolutely without result in non-susceptible persons. These symptoms are to be produced not only by local external application of pollen toxin, but hypodermic injection of very small quantities produces the most severe results—conjunctivitis, coryza, cough, asthmatic attacks, etc. The author reports a most remarkable case of this sort.

Following this discovery, the author began experiments directed toward obtaining a curative serum. Animals were injected with the pollen toxin. For months the serum not only did not neutralize pollen toxin, but rather increased its activity. Gradually, however, an antitoxin developed which completely neutralized the pollen toxin *in vitro*. More than that, it was possible on letting several drops of antitoxin fall into the eye or nose shortly after toxin had been previously applied, to entirely counteract its effect. This, however, has been as yet possible only in the very early stages of the manifestations. It is, however, much to be hoped that an antitoxin sufficiently strong to prove of actual therapeutic value may soon be obtained.

The important question naturally arises as to whether the toxin in all these different varieties of pollen is the same substance. Dunbar has already examined eighteen different varieties of pollen. He has already shown that the toxin of rye pollen granules is fully neutralized *in vitro* by the corn pollen antitoxin, while the symptoms produced by rye pollen toxin in a hay fever patient could be immediately arrested by corn pollen antitoxin. These observations would lead one to believe that pollen toxin of the various gramineæ is the same substance.

Juvenile Tabes and Hereditary Syphilis.—Linser (*Münch. med. Wochenschrift*, 1903, I. p. 637) reports the case of a woman, a virgin, who had suffered during childhood from frequent cutaneous eruptions, the nature of which was uncertain, as well as with glandular swellings, and presented, at the age of thirty-four, a distinct picture of locomotor ataxia. The first symptoms, of headache and dizziness, began at her fifteenth year; lancinating pains appeared at twenty, while at twenty-five the loss of light and patellar reflexes as well as paræsthesiæ were evident.

Until ten or fifteen years ago the existence of these juvenile cases of tabes was doubted by many. Twenty-one such cases have, however, been reported,

In about two-thirds of these cases ataxia was absent, though the loss of pupillary reflex, Westphal's and Romberg's symptoms, sensory disturbances, and lancinating pains were almost always noted, and often bladder symptoms and optic atrophy.

In most cases the symptoms began at puberty. An extremely slow and gradual progress of the symptoms is characteristic; long intermissions in the course are the rule. There is every reason to believe that *hereditary* syphilis is the main, if not the only, cause of this form of tabes. In seventeen of the twenty-one cases of juvenile tabes, syphilis occurred in one or other of the parents; in two it was probable; in two only questionable. In Linser's case the father acquired syphilis the year before the birth of the patient and died later of general paralysis.

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

JOHN RHEA BARTON PROFESSOR OF SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE UNIVERSITY HOSPITAL,

AND

F. D. PATTERSON, M.D.,

SURGEON TO THE DISPENSARY OF ST. AGNES' HOSPITAL; ASSISTANT SURGEON TO THE DISPENSARY OF THE UNIVERSITY HOSPITAL.

Hard Traumatic Œdema of the Back of the Hand and Foot.—VULLIET (*Centralblatt für Chirurgie*, 1902, No. 42) states that it is perhaps of interest to consider this rare condition, which has never been previously described, and which by its sequelæ becomes a morbid condition of importance. Until this time this condition has only been generally mentioned as one of the diseases of the hand, but its signs and symptoms are so characteristic that they deserve to be specifically mentioned. Briefly the condition may be considered as follows: (1) Etiology, usually the result of a blow upon the hand or foot; more rarely dorsal flexure of the hand may be the cause. (2) Symptoms: At first a diffuse swelling of the hand or foot, more pronounced at the base of the fingers or toes. At the height of the disease this swelling is hard and elastic and does not pit upon pressure. The back of the hand resembles an elastic cushion, without wrinkling of the skin, inflammation, or ecchymosis. At points a sense of crepitation may be detected, and the thenar eminence appears normal. Examination with the X-rays shows the bones to be uninjured, the parts are painful on pressure, and motion of the fingers is painful and restricted, except the thumb, which is unaffected. (3) This disease runs a very prolonged course, the swelling remaining the same for some time, and then grows slowly less, and after a period of from eight to twelve weeks it disappears. Sometimes it lasts longer, and then one finds either a hard, well-defined knot or a diffuse, vague hardening over the

bones which rarely is permanent. A return to work should not be made while the swelling persists. (4) The pathology has never been directly studied, but theoretically there is a diffuse, fibrous exudation under the skin, which is either slowly absorbed or becomes organized. (5) The differential diagnosis from the œdema of inflammation is easy, as the latter is soft and red, with a wound at the point of infection and involvement of the neighboring lymph glands. The differentiation from œdema, due to disturbed circulation as the result of pressure, is also a simple matter. Fracture of the metacarpal bones gives more localized symptoms with ecchymosis, the duration of the swelling is shorter, union is followed by deformity due to callus formation, and an X-ray examination makes the diagnosis positive. Hemorrhage is followed by ecchymoses, which are always absent in traumatic œdema. As a rule, the diagnosis is easy, but it can become a matter of difficulty when the hard and diffuse œdema lasts a long while, or when it becomes localized over a metacarpal bone. In all cases one should remember the traumatic origin of the disease, and not confuse it with a chronic inflammation, such as tuberculosis. (6) The treatment should consist of warm baths. Massage has proven more harmful than beneficial, and pressure bandages proved to be perfectly useless.

Extradural Hemorrhage from Rupture of the Middle Meningeal Artery.—JOPSON (*Annals of Surgery*, March, 1903) reports in detail three cases. In the last one the symptoms closely simulated those of extradural hemorrhage, but which operation showed it to be absent. In the first case the line of fracture crossed the anterior branch of the artery and explained its laceration. In the second case there was no fracture; the contusion was sufficient to rupture the vessel, a possibility well recognized. As regards the symptomatology of these cases, in the first one the classical clinical picture of extradural hemorrhage was absent, the symptoms being more those of fractures of the skull, with concussion or injury of the brain. The leakage of blood through the fissure into the subaponeurotic space probably relieved the pressure on the brain for a time and accounted for the preservation of consciousness and absence of paralysis. The temperature, slightly subnormal at first, then moderately elevated, was most suggestive of hemorrhage. The slow pulse is not always observed with epidural clot, but in this case the sudden drop to 48 before operation was significant of cerebral compression. In the second case the clinical picture was most complete. The initial stunning, the interval of consciousness followed by rapidly deepening coma, with subnormal temperature, slow pulse, stertorous breathing and contralateral hemiplegia, with dilatation of the pupil on the affected side, made up a symptom complex which scarcely admitted of other interpretation. In the last case the diagnosis of hemorrhage, while less certain, was justifiable, and trephining strongly indicated. There was a history of loss of consciousness, followed by its partial restoration, and then in a short time a deepening coma, with severe convulsions, starting on the opposite side from the injury, and followed by paralysis of the face on that side. The exact nature of the lesion remains unknown, but it seems probable that there was a slight hemorrhage or laceration in the motor area. The measurement used for locating the trephine openings and exposing the anterior branch of the artery in the

last two cases—two finger-breadths behind the external angular process of the frontal bone and three finger-breadths above the zygoma—corresponds very closely to the measurement formerly recommended by Treves, viz., one and one-half inches behind the external angular process of the frontal bone, and one and three-quarter inches above the zygoma. Treves now recommends a point located on a line drawn through the supra orbital ridge parallel to Reid's base line at a point from 3 to 4 cm. behind the external angular process. Pluminet has found all the measurements recommended for exposing the posterior branch to be unreliable, but Steiner's as open to the least objection. This locates it at a point where a line drawn horizontally through the glabella is intersected by a vertical line running just in front of the mastoid process.

The Technique of Lavage of the Stomach.—NECK (*Centralblatt für Chirurgie*, December 27, 1902) states that in washing out the stomach the question at this time is the danger attending the outflow or siphonage of the contents. The usual procedure consists in the introduction of a tube into the stomach; then the fluid is poured into the stomach through this tube, and then siphoned off again. In many cases this flowing out goes on easily, and apparently all of the fluid introduced comes away, but sometimes there are difficulties. Occasionally it is quite impossible to get all of the fluid to flow out of the stomach, even though a large amount of solution be put in. Even moving the tube about and the consequent straining on the part of the patient fail to empty the stomach. Such difficulty is often encountered in the cases where there is a motor insufficiency of the organ, especially when complicated by gastrectasis and pyloric stenosis. The question of completely emptying the stomach of its contents is an important one before operating upon that organ, as any fluid that may be present at the time the stomach is opened only tends to complicate the operation. In order to avoid this complication the author has endeavored to find a safe and easy method by which the stomach may be completely emptied of its contents. In the usual way of practising lavage the patient is placed in a horizontal position, or else remains sitting up. The author states that he believes in retaining either of these positions only as long as the siphonage goes on freely, and then the patient should be placed in the full pelvic position, and this will be followed in nearly every case by a return of the flow. The tube should then be slowly withdrawn (while the patient remains in this position), and as it comes out the last of the stomach contents will be withdrawn In each case in which this method was practised the stomach was found to be completely empty at the time of operation. The author has also experimented extensively on the cadaver with this method, and in each instance with great success. In conclusion he states that in those cases where the stomach is filled with particles of food the lavage must be repeated several times before the organ is empty.

The Diagnosis of Intestinal Injury following Abdominal Contusion.—LE CONTE (*Annals of Surgery*, April, 1903), after giving the history and treatment of thirteen cases, states in conclusion: First. That a moderately assured diagnosis of grave injury must be made before operation is undertaken, or one will open many abdomens to find the trauma confined to the

abdominal wall. In a series of 100 consecutive cases of abdominal contusion as they enter a general hospital, perhaps 30 or 40 will have received a grave injury demanding operation, while the other 60 or 70 recover without any operative procedure. For the sake of argument, the author states that he is willing to grant that if the abdomen is immediately opened in each one of the 100 cases there will result a smaller percentage of deaths than if the surgeon waits for some other symptoms of intestinal damage. But can one call such radical and empirical treatment the science of surgery? Would any one receiving a blow on the stomach sufficient to shock and nauseate say, "Have Dr. —— see me, for I want my abdomen opened at once?" Answering for himself, the author says No, for he should wish the surgeon in attendance to be moderately assured of his diagnosis before taking that smallest of risks, viz., an abdominal section in the hands of the most skilful surgeon. The author states that if he were one who always, with one exception, advocated immediate operation in appendicitis as soon as the diagnosis is made, he could with greater force urge immediate operation in all cases of abdominal contusion, for the seriousness of the two conditions is scarcely to be compared. The teaching of many of the modern writers when they urge operation in all cases presenting pain, rigidity, and local tenderness, seems too radical, for there are various kinds of pain and tenderness and different degrees of rigidity, and many times these symptoms are due to injury of the abdominal wall alone. Had the author followed such teaching, he states, he would have opened the abdomen in five of the reported cases, each of which presented pain, localized tenderness, and rigidity, and yet they all recovered without any exploratory operation. One should wait for some symptom or symptoms indicative of intestinal injury. Second. In the presence of shock, one cannot make a diagnosis of intestinal injury, no matter how profound the shock may be or how slowly reaction takes place. One may diagnose hemorrhage, which would lead to an immediate operation, and at the same time presume the presence of a lacerated gut, but primary shock is of itself no aid to diagnosis. One, therefore, should wait for reaction to take place. Third. No one symptom is pathognomonic of intestinal injury, but the two most reliable are gradually increasing rigidity and facial expression. In the next group the author places deep and, perhaps, radiating abdominal pain; respiration, which becomes more and more thoracic; vomiting after the shock has ceased; distention; increased pulse-rate, and secondary fall in temperature. The order in which they have been mentioned has no significance, for any one or two of these symptoms may be prominent to the exclusion of others. Fourth. Any individual who has received an abdominal contusion sufficiently severe to call for professional services demands also the most careful and constant watching in order that one may detect at the earliest possible moment the appearance of grave symptoms. This does not mean that one should wait for these symptoms to become so pronounced that a positive diagnosis is assured, for then operation is for the most part too late. There is a position, however, midway between operating on every case and waiting for an assured diagnosis, where one can say that, owing to the gradual appearance of certain symptoms, one has fair reasons to think the intestinal tract may be injured, and that under such circumstances an immediate operation will give the patient the best chance. In such a case one must not

forget the possibility of perforation taking place hours or even days after the injury. Lastly. As one's individual experience increases, one gains the power to place a more just value upon the symptoms present and to perceive the grave symptoms in their early stages. In other words, there is a gain in acuteness of perception, and there is scarcely any injury to the body which requires this more for a successful result.

THERAPEUTICS

UNDER THE CHARGE OF

REYNOLD WEBB WILCOX, M.D., LL.D.,

PROFESSOR OF MEDICINE AND THERAPEUTICS AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL; VISITING PHYSICIAN TO ST. MARK'S HOSPITAL,

AND

SMITH ELY JELLIFFE, M.D., PH.D.,

PROFESSOR OF PHARMACOGNOSY AT THE COLLEGE OF PHARMACY; CLINICAL ASSISTANT AT THE VANDERBILT CLINIC (COLUMBIA UNIVERSITY), NEW YORK.

The Use of Colloidal Silver in Infectious Diseases.—DR. NETTER enumerates a number of infectious diseases that have been promptly relieved by the use of colloidal silver or collargol, which was introduced as a therapeutic agent in 1897 by Credé. It is an allotropic form of silver which is soluble in water and in the secretions of the body. Colloidal silver is used chiefly for external applications in the form of a salve, 15 to 100 (*Unguentum Credé*), and in intravenous injections in a solution of 1 to 200. The amount of salve used in an application is 15 to 45 grains, according to whether the patient is a child, a youth, or an adult. For intravenous injections the amount varies from one-half to five-sixths of a grain. The use of collargol has grown widely, and many physicians report remarkable cures in many different diseases. Wenchebach cites two cases of severe infectious endocarditis that were cured by means of the injections, while Klotz obtained wonderful results in a septic endocarditis. The author reports ten different kinds of cases in which he had excellent effects from this remedy, among which was a case of pneumonia in which, after intravenous injections, the fever dropped immediately and disappeared entirely in three days. A suppurating cerebrospinal meningitis was modified as suddenly, apyrexia the day after the injections, and a rapid convalescence. The results were no less satisfactory in a severe scarlatina, two toxic diphtheritic anginas, and in three adynamic typhoids. The last observation was of a medico-surgical nature. A physician seized with pyohæmia found almost instant relief after a single application of collargol. Both the patient and the nurse who made the application noticed a distinct metallic taste. The return of strength, appetite, and ability to move came the following day. It is not certain that collargol acts antiseptically. Perhaps there is a neutralization of the toxins which produce such rapid changes in the body, and perhaps there is stimulation of the defensive power of the organism, or a catalytic action.—*Journal de Médecine*, 1903, vol. xv. p. 14.

Eukinase and Pancreatokinase.—MM. HALLION and CARRION describe the therapeutic application of two new drugs, discovered by Pawlof and his disciple, Schepowolnikow; the one, eukinase, is an intestinal eupeptic and the other, pancreatokinase, is a digestive ferment. It is easy to reproduce with eukinase the experiment that shows its value as a new drug. In two test-tubes, in each of which are placed equal portions of the cooked white of egg, there is added to the one a little pure pancreatic juice from a healthy animal, to the other an equal quantity of pancreatic juice and also a trace of eukinase. Under like conditions of temperature, etc., the white of egg in the first tube is digested very slowly, if at all, but in the second rapidly and completely. The practical experiments of physiologists bear out the results of those in the laboratory. In many intestinal affections, whether acute or chronic, or the result of infectious diseases, such a remedy could be used. Often such dyspepsias are confounded with gastric dyspepsias. Whenever the cause is in the non-efficacious action of the pancreatic juice, eukinase will prove a most rational intestinal eupeptic, as it permits and facilitates the action of the pancreatic juice. The association of pancreatin with eukinase makes a powerful digestive, which has been called pancreatokinase. To avoid the action of the gastric juice on eukinase, it has been prepared pharmaceutically in two forms, which are proof against the gastric juice, or else the active principle may be enclosed in a gelatin capsule, in which form it is easy to give it to children.—*Les Nouveaux Remèdes*, 1903, vol. xix. p. 25.

The Cure of Cholelithiasis by Means of Chologen.—DR. ROBERT GLASER describes the results of the treatment of a hundred cases of gallstone disease, in which he used a new preparation called chologen. He gives certain reasons which lead him to believe that cholelithiasis is a nervous and not an infectious disease. Upon this theory he bases his method of treatment. His chologen consists of three different organic quicksilver preparations, which he gives in tablet form and combines variously, making a preponderance of mercury, or of the aromatic vegetable element, as the character of the case may demand. In 73 women and 27 men whom he treated with chologen he found that 78 per cent. were cured; that is to say, they have been well ever since the treatment, having had no return of the attack, and been able to work and to digest without discomfort.—*Correspondenz-blatt f. den Schweitzer Aerzte*, 1903, vol. xxxiii. p. 73.

Enteroclysis in Acute and Chronic Dysentery.—DR. V. BELLEI makes some excellent suggestions concerning enteroclysis in Egyptian colic and the acute dysenteries. A non-irritating enteroclysis is one of carbolic water, 20 to 40 drops of the acid in a quart of warm water. The anæsthetic action of the carbolic acid tends to greatly assuage the colic and tenesmus, and it is so rapid and constant in its effect that this treatment may be considered almost a specific for Egyptian cholera. In chronic cases it is necessary to substitute the disinfectant enteroclysis by an astringent one to diminish the glandular and mucous irritation; and to continue them for several months. An interruption is apt to be followed by a violent attack, presumably due to reinfection of the mucous membrane; but the continued use of such lavage is apt to be poorly tolerated unless one avoids difficulty in the following way: (1)

By employing a mucilaginous liquid with an astringent one—two ounces of marshmallow-root to two quarts of water—forty-five to seventy-five grains of tannin, never more. The addition of thirty to forty drops of carbolic acid anæsthetizes the mucous membrane and helps considerably. (2) Tannin may be alternated with alum and alumnol, forty to sixty grains for two quarts. With this the attacks will be less frequent and less painful. (3) The most essential thing, without which enteroclysis cannot be borne well for more than two or three days, is to give simultaneously by the mouth thirty to forty-five grains of bismuth salicylate each day. It may be alternated with dermatol or tannigen to avoid a habit. Opium should never be employed.—*Revue de Thérapeutique*, 1903, vol. lxx. p. 80.

Cataphoresis in Gout.—Dr. Charles Begg claims to have obtained excellent results in the treatment of chronic gout and rheumatoid arthritis by this procedure. Two methods may be employed, either the joint is immersed in a solution of the drug to be employed, the positive electrode being placed in the bath and the negative on an indifferent part of the body, or the positive electrode is kept as wet as possible by frequent applications of the solution. The author employs a large negative electrode and a small button-shaped positive electrode.—*Edinburgh Medical Journal*, 1902, vol. vi. p. 547.

Acute Lead Poisoning in an Infant.—Dr. H. C. Cadman reports an interesting instance of lead poisoning in an infant five weeks of age. The symptoms were those of indigestion, with colic, the abdomen was hard and swollen. The face was of a peculiar gray color, and the child seemed dying. On close examination it was found that the mother was using a metallic nipple shield that was made of lead, and that the milk about the nipple had dissolved small quantities of it, and this was the cause of the poisoning.—*The Lancet*, 1902, No. 4135, p. 1458.

Theocin.—Dr. Chevalier has made an elaborate and complete review of the various literature concerning a new alkaloid, prepared synthetically, theocin, from the chemical, physiological, pharmaceutical, and experimental standpoint. Traube, a chemist, succeeded recently in manufacturing, in a practical and not too costly way, synthetic theopyllin, which is so difficult to extract from the natural product, tea, which has been introduced in its available therapeutic form under the name of theocin, a soft, white, bitter powder. The quantity of water eliminated by a healthy subject is considerably increased after the absorption of seven and one-half grains theocin. The quantity of mineral salts excreted is equally increased. It does not, however, seem to have any decided action on the elimination of the organic substances of the urine. Its efficacy has been established in cases where there was considerable œdema. There has, as yet, been no proof of irritation of the kidneys from the use of this substance. In cases where there had been albumin previously, it was not increased in proportion to the increase of the diuresis, and in other cases more has been caused. The therapeutic dose does not seem to influence the cardiac function. The frequency of the pulse and pressure of the blood are not at all modified. Its administration may

be attended at first by slight stomachic difficulty, such as loss of appetite and vomiting. It also, sometimes, has an exciting effect on nervous patients. To remedy these inconveniences it may be given in an alkaline combination or with hedonal. The doses in practical use are from three to seven grains in a cup of tea three times a day after each meal. A dose of seven and one-half grains, as in caffeine, should be considered a maximum.—*Revue de Thérapeutique*, 1903, vol. lxx. p. 73.

OBSTETRICS.

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.,

PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE; PROFESSOR OF OBSTETRICS AND DISEASES OF INFANCY IN THE PHILADELPHIA POLYCLINIC; CLINICAL PROFESSOR OF DISEASES OF CHILDREN IN THE WOMAN'S MEDICAL COLLEGE; VISITING OBSTETRICIAN TO THE PHILADELPHIA HOSPITAL, ETC.

The Disinfection of the Hands and the Occurrence of Puerperal Septic Infection.—No subject is of greater interest than the prevention of puerperal septic infection, and in no subject is it more difficult to form conclusions from the mass of conflicting testimony given by various investigators.

The use of rubber gloves seemed likely to exert a most favorable influence upon puerperal morbidity and mortality. In the *Zeitschrift für Geburtshulfe und Gynäkologie*, Band 45, Heft 3, 1901, STICHER, from the clinic at Breslau, reports the result of his observations in 1200 cases, including labor at full term, abortion, and the different operations which these conditions sometimes render necessary. The results of his investigations may be summarized by saying that the use of rubber gloves did not appreciably reduce puerperal septic morbidity nor puerperal septic mortality. In spontaneous labor with the use of rubber gloves the morbidity was from 10.5 to 12.1 per cent. His most fatal case of septic mortality occurred in a patient not examined by any method, and hence without the possibility of external infection in the clinic. In the manual removal of the placenta the results with the use of gloves were not so good as those without their use. Sticher does not believe that any precaution should be relaxed in the cleaning of the hands or in the use of gloves whenever possible. With strict disinfection of the hands, aided by gloves, it is possible to reduce puerperal morbidity and mortality of septic infection to the lowest point. It is, however, of the utmost importance that labor should be so conducted that wounds of the genital tract be avoided as much as possible, and that the retention of membranes or placenta in the uterus should not occur. The trend of his study is to lay great stress upon the disinfection of the external genital organs and the avoidance of the introduction of contagion by contact with them. He also believes that under some conditions the patient's genital tract may contain the elements of infection, irrespective of examination.

A more recent paper than that of Sticher is contributed by Schumacher, from the clinic at Strassburg (*Archiv für Gynäkologie*, Band 68, Heft 2, 1903). Schumacher made bacteriological examinations of the hands of physicians, students, and midwives, and found that when the hands were well disinfected streptococci were not obtained from the integument. Lower orders of bacteria, however, were obtained from the hands, but their number was greatly reduced and also their virulence by disinfection. In fact, it was the rule, and not the exception, to obtain these less virulent bacteria from the hands. Much the same results were obtained in the clinics at Halle on the hands of persons who performed or assisted at a series of cases of abdominal section. It was curiously observed that it is easier to make the right hand free from bacteria than the left.

A comparison was then made between the number of bacteria found on the hands and the puerperal morbidity. For this purpose a series of 115 labors was investigated, with a morbidity of 17.3 per cent. It was found that in those cases in which the hands showed the presence of the yellow pus-producing bacteria, puerperal morbidity was greatest. When the hands and arms were found to be free from bacteria, the puerperal morbidity fell to 10.5 per cent. When the hands contained abundant bacteria, it rose to 15 per cent. When, however, the hands were examined in cases that had no morbidity, the surprising result was obtained that in cases proceeding normally the hands frequently contained more of the staphylococcus aureus than in those cases that had fever. The result of these studies was the failure to establish a relationship between the number of colonies of staphylococci obtained from the hands and the presence or absence of puerperal morbidity. While we cannot render the hands free from all bacteria, we do remove streptococci by faithful cleansing, and we cannot prove that the remaining germs markedly influence puerperal morbidity and mortality.

When hands covered with rubber gloves were examined, it was found that it was possible to remove bacteria in most cases from the surface of the gloves. In some cases, however, the less virulent forms of bacteria were still present. The use of gloves, however, was followed by a very slight difference in puerperal morbidity and mortality. So far as the use of gloves in examinations was concerned, it could not be ascertained that their use occasioned a distinct improvement. Those bacteria which remained upon the hands after faithful disinfection had no influence upon the puerperal state. For normal labors and for the purpose of teaching, Schumacher did not find that the use of rubber gloves should be recommended. When, however, the hand has been recently infected, or when there is reason to fear infection of the hand, then the use of gloves is alike proper for the defence of the patient and also the physician.

Stolz, of Graz, has recently published a book of over 300 pages, entitled *Studies in the Bacteriology of the Genital Canal during Pregnancy and the Puerperal State*. This book bears directly upon the question under discussion.

He found in the secretion of the vagina pathogenic bacteria in normal and pathological cases. The so-called destructive influence of the vaginal secretion, he believes, has not been proven. Likewise in the puerperal state he found but 50 per cent. of lochial discharges practically sterile, and does not

believe that the uterus in normal cases is germ free. His technique was as thorough as possible and after approved methods. He did not find that repeated examinations during labor, the length of labor, the length of the third stage, lacerations of the perineum, and operations done with sterilized hands played any essential part in increasing the bacteria found in the lochial discharges. The one feature which greatly increased the bacterial contents of the lochia was retention of the membranes.

In patients having an afebrile puerperal period the lochia contained bacteria in 80 per cent. and streptococci in 36 per cent., the increase in germs taking place soon after the patient's delivery. When fever occurred in most cases the uterus succeeded in cleansing itself from bacteria. The streptococcus was most frequently present in these cases. In 15 per cent. the uterus relieved itself spontaneously of the infective agent. Intra-uterine douches of 1 per cent. lysol were useful, especially in removing retained membranes or blood-clot.

The use of rubber gloves did not materially alter the results previously stated. A case of fatal infection occurred in a patient examined with the use of rubber gloves only. Operations performed by hands covered with sterile gloves gave no better results than those done with disinfected hands. Only in the case of very frequent examinations did the use of the gloves seem to be an advantage. The result of this book is to emphasize the possibility of autoinfection by the ascent and absorption of bacteria already within the vagina at the termination of pregnancy.

Two Cases of Lateral Section of the Pelvis by Gigli's Method.—PESTALOZZA (*Centralblatt für Gynäkologie*, No. 4, 1903) reports two cases in which he practised this operation in his clinic at Florence.

The first case was a woman in her seventh pregnancy, who had lost children from birth pressure in previous labors. She had a symmetrically contracted pelvis, whose internal anteroposterior diameter was between 8 and 9 cm. The child was large. The patient had had strong pains, but the head remained movable above the pelvic brim. On cutting through the skin slight hemorrhage occurred, as the incision was carried a little too far down and too near the vulva. This was readily controlled. The bone was very easily separated by a chain saw and the child readily delivered by forceps. The wound was closed without drainage and union occurred by first intention, the temperature remaining normal. The pelvis completely united. The child weighed 3850 grammes, and nursed well until the twentieth day. It then died suddenly in convulsions. Upon autopsy a slight icterus and slight œdema of the brain were the only lesions found.

His second case was in her third pregnancy, and had had two small children in spontaneous labor. Several attempts were made to bring the head down by forceps, but without success. After section of the left pubic bone, a living child, weighing 3800 grammes, was readily delivered. The wound was closed by deep sutures passing through the fascia and periosteum. There was no drainage. The patient's temperature rose to 100° on the second day, but speedily fell. Mother and child made a good recovery.

In comparing this operation with symphysiotomy, the writer draws attention to the fact that it would be possible to wound the bladder. There is no

especial danger of this, however, if the operator exercises reasonable caution. In these cases the urine was bloody for a short time after the operation without assignable cause. A catheter was placed in the bladder during the operation to hold the urethra out of the way.

GYNECOLOGY.

UNDER THE CHARGE OF

HENRY C. COE, M.D.,
OF NEW YORK.

ASSISTED BY

WILLIAM E. STUDDIFORD, M.D.

Colon Bacteria in the Peritoneal Cavity.—SOLIERE (*Ziegler's Beiträge zur path. Anat.*, Band xxxi.), after reviewing the literature of the subject, reports the results of his experiments with guinea-pigs, in which he aimed to increase the power of resistance of the healthy peritoneum to infection by injecting into it a sterile solution of saline, fresh broth, and sarcolin. A drop of fluid containing colon bacilli was then introduced into the cavity through a hypodermic syringe. Beginning with the smallest fatal dose the writer was able to gradually increase it up to twenty-three times this strength, by giving a prophylactic injection of the before-mentioned solution the day before. It was thus proved that the power of resistance of the peritoneum could be increased from seven to sixteen fold.

BIONDI conducted the same experiments in three human subjects, using prophylactic injections of from 30 to 60 c.c. of normal salt solution. In two instances the subsequent removal of ascitic fluid allowed precise conclusions to be drawn with regard to the presence of the bacteria. In spite of the use of these preventive measures, there may be so many bacteria present that the peritoneum cannot withstand them, and death occurs speedily, or a less rapid fatal result may follow from the setting free of toxins from the phagocytes.

Disadvantages of Ventrofixation.—GRADENWITZ (*Zeitschrift für Gynäkologie*, 1903, No. 5) summarizes a paper on this subject, as follows: Suture of the stumps to the abdominal wall after removal of the adnexa is unnecessary, if continuous suture of the broad ligament is practised instead of transfixion and ligation. Ventrofixation by suture of the round ligaments without removal of the adnexa, exposes the patient to the dangers resulting from the formation of pockets; a better result can be obtained by Alexander's operation. Fixation of the fundus uteri offers the most permanent relief for retroflexion, but should be rejected on account of the danger of metritis, hernia, and subsequent disturbances attending pregnancy and parturition. Shortening of the round ligaments or vaginal fixation is preferable.

Thrombosis following Abdominal Section.—RIEDEL (*Centralblatt für Gynäkologie*, 1903, No. 3) reports eight cases in which the thrombosis was on the left side, and three on the right. Two of the latter followed fixation of the right kidney. The left vein was affected in five cases of appendicitis, in one of which no operation was performed. The thrombosis in most instances did not develop until several weeks after operation, and the majority of the cases were aseptic (?).

The writer attributes the complication to the prolonged recumbent posture, and explains its greater frequency in the left vein as due to the pressure of the superimposed arteries, which cross it at a more obtuse angle than on the right.

Adhesions following Laparotomy.—PEMHORST (Inaugural Dis.; abstract in *Centralblatt für Gynäkologie*, 1903, No. 6) studied sixteen cases in which the condition of the abdomen was noted at the time of a secondary operation or at autopsy. In all a previous abdominal section had been performed, and the most careful means had been employed to prevent the formation of adhesions by covering the stumps and raw surfaces with flaps of peritoneum, making as small an incision as possible to avoid the entrance of air and suturing the peritoneal edges exactly, and filling the bladder before closing the cavity.

In only four cases were adhesions absent, mostly between the omentum and the abdominal wound. In three cases they were slight, in four extensive, in three there were adhesions of the intestines to the stump. If there was elevation of temperature after operation they were always found, but in three cases the convalescence had been afebrile. The writer concludes that adhesions are the result of local inflammation, and that they cannot be prevented by the most rigid aseptic precautions.

Results of Hysterectomy for Cancer of the Uterus.—GLOCKNER (*Centralblatt für Gynäkologie*, 1902, No. 52) reports the results of operations at the Leipzig Clinic between 1887 and 1901. Nine hundred and seventy-four cases of cancer of the uterus were treated, 260 of which were regarded as suitable for radical operation. Vaginal hysterectomy was performed 225 times, 4 patients were subjected to cœliotomy, and in 24 the combined method was adopted. The peritoneum was sutured in 115, and the cavity was left open in 139; 22 died, or 8.46 per cent. Injuries to the uterus, bladder, or intestines ocurred 18 times, 4 being fatal.

One hundred and sixty-two cases were kept under observation for five years, of which 47 (35.6 per cent.) remained free from recurrence.

Etiology of Tubal Pregnancy.—OPITZ (*Zeitschrift für Geb. u. Gyn.*, Band xlviii., Heft 1), from a study of twenty-three specimens of tubal gestation, arrives at the conclusion that the cause of arrest of the impregnated ovum in the tube is the presence in the latter of cul-de-sac formed by the adhesion of neighboring folds of mucous membrane, the result of previous attacks of salpingitis. These false cavities were found in every specimen examined by examining numerous serial sections. This explanation has not been offered by any previous observer.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

EDWARD JACKSON, A.M., M.D.,
OF DENVER,

AND

T. B. SCHNEIDEMAN, A.M., M.D.,
PROFESSOR OF DISEASES OF THE EYE IN THE PHILADELPHIA POLYCLINIC.

Operation for Conical Cornea with the Galvanocautery.—DODD, London (*Medical Press and Circular*, 1903, No. 7), operated on a girl, aged nineteen years, affected with double conical cornea, with the galvanocautery, using a modified method of Critchett's operation. Critchett's method is to apply the cautery to the centre of the cornea until almost a concave depression is formed, taking care not to perforate into the anterior chamber. Other operations have been suggested, such as cutting off a small superficial flap of the cone and subsequently cauterizing the denuded surface; repeated paracentesis, to let out the contents of the anterior chamber to lessen the intraocular tension; removing a small disk from the apex of the cone with a specially made trephine; the excision of an oval piece of the cornea.

All of these operations result in a thick, central scar, requiring an iridectomy for visual purposes. To avoid the necessity of the latter, Dodd applies a small galvanocautery in a series of dots about 1 mm. apart, so as to form a horseshoe-shape around the cone, leaving the centre free. The operation may have to be repeated at intervals to get sufficient contraction.

Operation for conical cornea is the only treatment which holds out hope. The vision in these cases is extremely defective, and cannot, as a rule, be made satisfactory by any form of glass.

Plastic Artificial Vitreous in Mules' Operation.—OATMAN, Brooklyn (*Medical Record*, March 7, 1903), reports three cases of Mules' operation, in which paraffin instead of a glass ball was used as an artificial vitreous.

When from any cause a fistulous opening has once formed over an artificial vitreous it resists all attempts at repair until the foreign body has been expelled. Under these circumstances it seemed to the writer that if a portion of the ball could be removed and the tension on the scleral walls relieved closure of the wound over the remainder might be hoped for later. For this reason he selected paraffin for the artificial vitreous, so that, if necessary, it could be removed piecemeal.

Oatman draws the following conclusions from his case: When fistula follows the Mules operation it will close spontaneously if a sufficient quantity of the artificial vitreous can be removed. That which remains will form a good support for an artificial eye.

Paraffin used for this purpose is prone to produce fistula by softening and

getting between the lips of the wound or into the track of a suture. These accidents are to be avoided by using paraffin or some modification thereof that will not soften at body temperature; also by suturing the scleral wound with silk, so that no aperture remains through which paraffin can exude.

A plastic material like paraffin will adapt itself to any inequalities on the surface of the glass shell, and ulceration from pressure is not apt to occur.

An Astigmatism Cured by Tenotomy.—BULL, Paris (*New York Medical Journal*, 1903, No. 6), reports a case of myopic astigmatism of 1.75/D against the rule, shown by the ophthalmometer to be almost entirely corneal, which disappeared after complete tenotomy of the external rectus of that eye. The author remarks that the importance of the case depends not upon the cure of the astigmatism, but upon the light which it throws upon other questions, obviously that of the intraocular tension. Inverse astigmatism, sometimes progressive, so common in glaucoma, may find its explanation in the position and relative tension of the ocular muscles.

[Dr. Bull makes some just remarks upon the improper term *astigmatism*, for which he proposes the philologically correct form *astigmia*. The word astigmatism is derived from ἀ, privative, and στιγμα, genitive, ατος, a stain or prick, and this, of course, does not express our meaning. Astigmia comes from ἀ, privative, and στιγμη, genitive, ῆς, a mathematical point—just what we intend to say. He also suggests that the expressions "with the rule" and "against the rule" be replaced by the single terms "direct" and "inverse." These sensible suggestions should find ready acceptance.]

Treatment of Hypopyon Kerato-iritis.—BURNHAM, Toronto (*Lancet*, December 6, 1902), advocates purely constitutional treatment of this condition, dispensing with all local interference save atropine once every day or second day and bathing with hot water or boric acid solution. The internal remedies are the combined treatment—mercury and iodide of potash by the mouth, and pilocarpine hypodermically. He claims for this plan rapid relief from pain and general improvement, no relapses, and, finally, great diminution in the cicatricial opacity.

Treatment of Trachoma by the High-frequency Current.—DR. WALSH, London (*Medical Press and Circular*, 1903, No. 7), has had an extremely favorable result in treating a single case by the high-frequency X-ray current. Apparent cure was, in fact, obtained after twenty-two applications. The applications were made to the uneverted lids. The period of exposure ranged from eight to fifteen minutes; generally the latter.

Amaurotic Cat's Eye.—SCHEIDEMANN (*Berliner klin. Wochenschrift*, 1903, No. 2) describes two enucleated eyeballs which had exhibited the appearances of the amaurotic cat's eye, but were very different pathologically: the one was a case of glioma, the other an intraocular cysticercus. The author emphasizes the favorable results of early enucleation in glioma, and calls attention to the occasional occurrence of sympathetic irritation of the other eye in cysticercus. The latter disease has become rare in Germany since the governmental inspection of meat.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
SIMON FLEXNER, M.D.,
PROFESSOR OF PATHOLOGY, UNIVERSITY OF PENNSYLVANIA.
ASSISTED BY
WARFIELD T. LONGCOPE, M.D.,
RESIDENT PATHOLOGIST, PENNSYLVANIA HOSPITAL,
AND
F. P. GAY, M.D.,
ASSISTANT DEMONSTRATOR OF PATHOLOGY, UNIVERSITY OF PENNSYLVANIA.

Further Communications upon the Pathogenesis of Icterus.—EPPINGER (*Beiträge zur Path. Anat. u. z. All. Path.*, 1903, vol. xxxiii. p. 123) in the present communication extends his former observations made upon the purely mechanical forms of icterus to a consideration of other varieties of this affection. The author now states that in the typical cirrhoses of Laennec, certain of the bile capillaries or so-called precapillaries are much constricted or even completely closed by the new connective tissue growth in the portal spaces of the liver. As a result there ensues a dilatation of the bile capillaries with ultimate rupture and escape of bile into the perivascular lymphatic spaces. A study of the liver from cases of phosphorus poisoning, representing the type of toxic jaundice, showed that the trabecular bile ducts were occluded by small thrombi. This led to stasis in the intercellular bile capillaries with subsequent widening and, as in the cirrhotic livers, to rupture of the capillary walls, followed by a pouring out of the bile into the perivascular lymph spaces. In still a third variety of jaundice, "the cyanotic icterus," following upon a chronic congestion, and due principally to cardiac disease, thrombi were again found occluding the bile capillaries. Thus the author explains the icterus accompanying cirrhosis of the liver as well as the jaundice of phosphorus poisoning and cyanosis, upon a mechanical obliteration of the finer bile ducts, with rupture of the ducts and escape of bile into the lymphatics.

The Influence of Nephrectomy upon Absorption (an experimental study from the Rockefeller Institute for Medical Research).—MELTZER and SALANT (*Journal of Medical Research*, 1903, vol. ix. p. 33), in a former study upon the effects of subminimal doses of strychnine upon nephrectomized rabbits, noted that such animals could receive much more than the fatal dose without manifesting any reaction. This result suggested that in nephrectomized animals absorption from the subcutaneous tissues might be decreased. In order to determine the power of absorption in animals in which the kidneys had been removed, rabbits were subjected to double nephrectomy, salt solution was introduced into the peritoneal cavity, and after a varying time the animals were killed, the peritoneal cavity opened, and the amount of fluid absorbed measured. Contrary to expectation, it was found that

with an 0.8 per cent. solution of sodium chloride absorption was as good and often better in nephrectomized animals than in normal animals. With a 1.2 per cent. solution of sodium chloride, which is decidedly hypertonic, the difference between absorption in normal and nephrectomized animals was even greater, absorption being decidedly greater for the nephrectomized animals. With 1.5 per cent. solutions, it was frequent to find an increase of fluid in normal rabbits, whereas this never occurred in nephrectomized animals. The increase of absorption for the nephrectomized rabbits continued to manifest itself even when solutions were introduced into the peritoneal cavity about twenty-four hours after nephrectomy. At a period of about forty hours, however, the factor favoring absorption began to diminish. When the fluids were left in the peritoneal cavity for five hours or longer, the rates of absorption in the normal and abnormal animals were about the same.

From these experiments the authors conclude that after nephrectomy in rabbits the osmotic pressure of the blood is increased, and therefore in these animals solutions of sodium chloride are readily absorbed from the peritoneal cavity. The osmotic pressure of the blood of nephrectomized animals appears to correspond to the osmotic pressure of a solution of sodium chloride, having a concentration somewhere between 1.2 per cent. and 1.5 per cent. This increased power of absorption following immediately upon removal of the kidneys seems to explain the absence of œdema after nephrectomy in animals, and after prolonged anuria in human beings. The first effect of anuria, according to the authors, is not an increase of lymph in the tissues, but rather an increase of absorption from the tissues into the circulation, thereby preventing œdema.

Eleven Acute and Eighteen Chronic Cases of Influenza Proved by Bacteriological Examination.—LORD (*Boston Medical and Surgical Journal*, 1902, vol. cxlvii. p. 662) has investigated the cause of cough and expectoration in 100 cases in which sputum examination proved negative for tubercle bacilli. In the sputum of 60 of these patients he found influenza bacilli. The sputum in 29 cases showed the presence of influenza bacilli in such overwhelming numbers that these organisms appeared to be the cause of the cough. In the other 31 instances in which influenza bacilli were found, they were mixed with pneumococci, staphylococci, and streptococci, and were considered as possible secondary invaders. Some of the cases were acute and others chronic, the latter being often of several years' duration. Many of the chronic cases were mistaken for chronic bronchitis, and a few in which paroxysmal dyspnœa was a prominent feature closely resembled asthma. The author concludes that, apart from an epidemic of influenza, infection of the respiratory tract with influenza bacilli is not uncommon.

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All communications should be addressed to

DR. FRANCIS R. PACKARD, 1831 Chestnut Street, Philadelphia, U. S. A.